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VETERINARY MEDICINE

MARCH CONTENTS 1949

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D. M. CAMPBELL, Editor
7632 S. Crandon Ave., Chicago 49, Ill.
Telephone SO uth Shore 8—7160

H. J. MOSTYN, Eastern Representative
P. O. Box 391, Dover, New Jersey
Telephone Dover 6-2711W.

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A look into the past unfetters the mind, gives a clearer conception of the present, and tends to strengthen our confidence in the future.

The Babcock method of determining the fat content of milk was invented in 1890.

The first bacterial count of market milk was made by Sedgwick and Batchelder in 1892.

In 1892 Theobald Smith isolated and identified the bacillus of bovine tuberculosis and determined the degree of heat that will destroy it.

The production of certified milk was first conceived by Coit of New York, but none was actually produced as a market commodity until 1900-1901, when a medical milk commission was established in New York City.

Galen (131-201 A. D.) recognized the importance of milk inspection. Having prescribed ass's milk for a patient he directed that the ass from which the milk was to be obtained should be inspected as to its general health.—*Smith's Early History of Veterinary Literature.*

Mrs. Nancy Johnson, the wife of an American naval officer, invented the modern ice cream freezer. It is said that the first ice cream served in America was at a White House reception during President Madison's Administration. From this departure Americans have developed an appetite for this confection that craves for 2,600,000,000 gallons a year.

Matriculation Requirement

A Regulation of the London Veterinary College [now Royal Veterinary College, London] in 1829 provided:

1. Any person desirous of becoming a pupil on the foundation of the college, must be able to read and to write legibly. Preference shall be given to those youths who shall have received the elements of a good education.

At that the course of study in the college was of 12 months duration, but many were given their diplomas after an attendance of less than six months. However conditions were on the mend and in the March (1829) issue of *The Veterinarian* we find:

"We stop the press, and somewhat inconveniently disarrange our matter, to state a rumor which has just reached us, and from a source on which we can place much reliance that the competent authorities at the Veterinary College have determined to enforce a longer residence at that school by every pupil.

"An attendance of at least 12 months, it is said, will be exacted from every student, although he may be the son of a farrier or of a veterinary surgeon.

"We announce this with pleasure and gratitude. The insufficient education of the veterinary surgeon has been the main cause of our lamentable degradation.

"This concession to public opinion is gratifying and honorable. We regard it as the harbinger of a new and happier era in the history of the veterinary art."

There is some evidence that penicillin prevents recurrence of rheumatic fever, following hemolytic streptococci infections.

Organo-therapy—the use of insulin, adrenalin, pituitrin, thyroid extract, liver extract, etc., depends upon the slaughter of animals at the larger packing houses. The small slaughterers do not have facilities for collecting and storing these glands for pharmaceutical manufacture. The pancreas, in particular, requires great care in aseptic collection, quick freezing and storage. During the "black market" slaughter near the close of the war, the entire reserve of insulin was exhausted and the country was on a day to day supply when conditions began to improve.

Bacitracin an Antibiotic Drug of Major Importance

Bacitracin, one of the newest trio of antibiotic agents with outstanding therapeutic qualities (the other two being chloromycetin and aureomycin) is effective against most strains of hemolytic streptococci, nonhemolytic streptococci, coagulase-positive staphylococci, pneumococci, gonococci, all of the gas gangrene group, *Clostridium tetani*, the actinomycotic group, the diphtheria bacillus, the pale spirochete, the anaerobic cocci in general and some protozoa.

Bacitracin has a synergistic action with penicillin and is effective against many strains of organisms that have acquired penicillin fastness. It has not been produced in pure or crystalline form as has penicillin and, given internally, it is somewhat toxic to the kidneys. Its principal use at present, both systemically and locally, is in conditions which have failed to respond to sulfa drugs or to other antibiotic agents.

On the whole the topical use of penicillin has been disappointing. Probably the principal cause of the frequent failure of penicillin as a dressing for infected wounds is that a large number of organisms contain a penicillinase which quickly destroys penicillin. Exudates often inactivate penicillin. So far as is known, bacitracin is inactivated by only one organism, and exudates have little effect on it. It is used in aqueous solution, as an ointment and in powder form in the treatment of wounds, boils, carbuncles, conjunctivitis and abscesses.

The naming of bacitracin was unusual. The bacteria from which it is derived were isolated from a bone infection in a little girl named Margaret Tracy. Thus the name combines bacillus, Tracy and the "in" ending common to all antibiotic agents of therapeutic efficiency.

The nutritional value of proteins depends on their amino-acid composition and grouping.

Meat scrap and tankage contain large amounts of niacin and are rich in riboflavin and pantothenic acid.

Only two of the amino acids contain sulfur—methionine and cystine. The former can substitute completely for the latter and is considered essential for animal growth.

The production of foot-and-mouth disease vaccine, begun in May 1948 by the joint Mexican-United States Commission reached the astounding total of 1,750,000 doses in December of that year.

The foot-and-mouth disease outbreak in Mexico cost the United States Government $44,939,232 to December 1948. In addition, to relieve the cattle surplus in northern Mexico occasioned by the border quarantine, the Commodity Credit Corporation purchased canned meat at a cost of $25,325,000 and has commitments to purchase $24,043,575 worth in 1949. Against this expenditure of 49 million dollars, the sum of $12,529,375 realized from the sale of canned meat, is to be credited.

Treatment of Tetanus

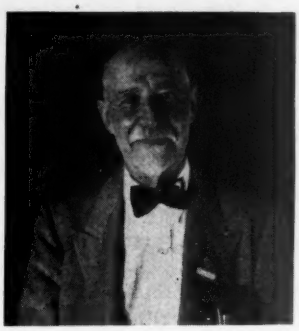
Couvy summarizes research of the French (*Bul.-Acad. Med.* III, 143) on the "blood brain barrier," represented by the meninges and the walls of the blood capillaries which supply the brain and other parts of the central nervous system. This barrier is impermeable to tetanus antitoxin as well as to a large number of medicinal agents.

A marked exception to this is urotropin, which flows to the central nervous system and, in addition, renders the barrier permeable to other agents, which otherwise are incapable of traversing it. This factor has been successfully used in the treatment of advanced cerebrospinal meningitis, nervous syphilis (which had entirely failed to respond to prolonged arsenical treatment). and human trypanosomiasis.

In the treatment of tetanus an injection of antiserum intravenously or subcutaneously is followed after 30 minutes by the intravenous or subcutaneous injection of urotropin, which in turn is followed after one-half hour by a second injection of antiserum.—*Vet. Rec.* 60:22, 1948. Abst. in *J. S. African Vet. Med. Assn.*

Veterinary Nestor Passes

Doctor Tyler died in his Whittier, California home December 24, 1948. He was a graduate of the Barnes Medical College, St. Louis and later of the Chicago Veterinary College in 1891. He began veterinary practice in Chebanse, Illinois, but found it hard going during the panic of 1893-94 when serviceable farm horses could be bought for $10. So he took up practice of medicine in Terre Haute, Indiana. But



JOHN LOW TYLER, M.D., D.V.S., 1870-1948

not for long as he soon engaged in veterinary practice in the rice section of Louisiana when because of financial reverses he was again forced to return to Terre Haute to resume medical practice. Failing health took him to California in 1897 where he again entered veterinary practice at Pomona, remaining there for more than 30 years but eventually retiring to Whittier, California where he lived at the time of his death.

Doctor Tyler was a frequent contributor to veterinary periodicals, often favoring this publication with up to 10 clinical reports a year. He was twice president of the Southern California veterinary association and president of the Los Angeles association a like number of times.

Veterinary Inspection In Japan

The veterinary section of the Military Government of Japan reported 372 cases of disease in farm animals in November. Bovine tuberculosis led in frequency of occurrence with 163 cases. There were 68 cases of equine infectious anemia. Six abortions due to Brucella and 94 due to trichomonads were reported. Cases of pullorum disease number 7483.

Cattle slaughtered numbered 22,664; horses, 5773; swine 18,764. Total condemnations were few, but condemnations of parts were very large.

Milk inspection included examination of a large number of samples; 6000 plant and 11,000 dairy farm inspections. Of the farms inspected 41 produced goat milk.

Retail meat shop inspection included more than 8000 establishments and a million kilograms of meat.

Responsibilities of Veterinary Medicine*

Should any one of a half dozen highly contagious diseases of cattle, swine or poultry be permitted to establish itself in this country and to spread unopposed, the consequences would be calamitous. It is the responsibility of veterinary medicine as the guardian of animal health to preclude such a catastrophe.

French Veterinary Schools

The first veterinary college of modern times was founded at Lyons, France in 1463 and the second in Alfort a suburb of Paris in 1765. The former soon rendered France such important services that Louis XV took it under his especial patronage.

Sixty-six years later the Royal Veterinary School of Toulouse was founded because:

"Two schools in France have furnished only a small number of veterinarians, scarcely sufficient to treat one-twentieth part of our animals."

Doctor McCulloch Dies After Long Illness

Doctor Ernest C. McCulloch died December 1, 1948 at Pullman, Washington. In his early years he was an Argentine salesman for an American firm. Stranded in Buenos Aires by failure of his employer he worked his way on a cattle boat to the U.S.A. and took up the study of Veterinary medicine at Kansas State College from which he received a degree of D.V.M. in 1924. He pursued graduate work at the University of Illinois, receiving an M.A. degree. He then studied at the University of Wisconsin for a Ph.D. degree in chemistry. Following this he taught bacteriology for a short time at the School of Veterinary Medicine at Auburn, Ala., going from there to research work on disinfectants and parasiticides for the Pennsylvania Salt Company. For a time before he joined the staff of the Washington State College in 1940, he had served as chief milk inspector for the St. Louis Board of Health. In 1945 the second edition of his book "Disinfection and Sterilization" was published. This book is the standard text on the subject. At the time of his death, Doctor McCulloch was professor of Bacteriology and Parasitology at Washington State College.

The dairyman depends upon hay, pasture, and other roughage for the vitamin A (carotene) indispensable to the reproduction and welfare of his cows, and only good roughage supplies this vitamin in adequate quantity for the lactating dairy cow or the rapidly growing calf.

Most plant poisoning in cattle is due to vitamin A deficiency. Poisonous plants are all, or nearly all, unpalatable to cattle and are seldom eaten when other green food is available. In the early spring, in the late summer and during long periods of drouth poison plants may be the only green vegetation in the cow pasture. Cattle that have been on an insufficient vitamin A diet for months will eat these green plants regardless of the taste.

Meat Scrap and Tankage

Meat meal or meal-scrap is the finely ground, dry rendered residue from animal tissue, exclusive of hair, hoof, horn, blood, manure, and stomach contents, except in such traces as might occur unavoidably in good factory practice.

Digester tankage, meat meal tankage or feeding tankage is the finely ground residue from animal tissues exclusive of hair, hoof, horn, manure, and stomach contents, except in such traces as might occur unavoidably in good factory practice, especially prepared for feeding purposes by tanking under live steam or by dry rendering or a mixture of the products.

"Meat and bone scrap" must not be used in connection with feed for dogs or fur animals.—*Nat'l Provisioner.*

Functions of Bulk in the Ration

All animals require a certain amount of bulk or roughage in the diet for normal digestive functions, but this requirement is much higher for the ruminant than for the non-ruminant. There is evidence to indicate that without a sufficient amount of roughage the bacterial action in the rumen of the dairy cow is disrupted. There are also indications that the quality or kind of roughage is also important in its influence on rumen functions. It is possible that this may be why the cow cannot survive indefinitely without roughage. It is conceivable, but not yet established, that the bacterial action in the rumen may be the only source of some of the essential factors for well being of the ruminant, and that when the roughage is lacking in quantity or quality, or both, the normal function of the rumen is disturbed.—(Purina) *Nutr. News Bul.*

The blood and urine of pregnant mares contains estrogenic and gonadotrophic hormones; the urine contains only estrogenic hormones.

Cows will live only about one year on grain alone (no roughage). They will live and remain in good health for many years on hay alone, according to E. B. Meigs, U.S.D.A.

The pellagragenic effect of corn, when it constitutes a large part of the ration, can be overcome by including niacin in the diet. Tankage is rich in this vitamin.

When designing a ration for dairy cows, more concentrate should be included when the roughage is poor than when it is good. Mainly because cows will eat 50% more of the good roughage than of the poor roughage.

To be relished by the dairy cow roughage must contain ample protein. In our experience we have never found roughage that was low in protein which would be liberally eaten by the dairy cow over a prolonged period.—(Purina) *Nutr. News Bul.*

Young dogs fed a niacin-deficient ration which contained 1% of sulfasuxidine developed anemia and black tongue. When given injections of niacin they improved two or three times then failed to respond to additional injections. When folic acid was added to the niacin improvement again resulted for two or three injections, then failed. When liver extracts rich in the antianemia factor was added to the niacin and folic acid and all three injected, recovery was spectacular.

Poultry waste is rapidly assuming a position of importance as a diet ingredient in the feeding of ranch-raised mink. Within the past two years many mink ranchers have begun using it as a substitute for horse meat in the rations for their fur animals. For various reasons horse meat, which has long been the standby for ranchers, is becoming a scarce and expensive product. The amount of poultry waste available for use in mink feed, on the other hand, should increase in the years to come, since poultry marketed in recent years has increased in both numbers and total weight. The total weight of heads, feet and viscera accumulating at a modern poultry dressing plant in a single day represents a vast amount of fur animal food if it is all utilized.—*Barrett and Welke. U. S. Fur Animal Exp. Sta.*

Comments Upon Animal Health Factors

By R. R. DYKSTRA, D.V.M., Manhattan, Kansas

It is reported from South Africa that a cattle tick, a strain of *Boophilus decoloratus*—the blue tick, appears to have developed resistance to dipping in a solution of benzine hexachloride.

For more than 100 years it has been known that cattle grazed on certain pastures in England contract "teart disease" which is characterized by anemia, diarrhea, loss of weight and decreased milk production. It is due to the presence of molybdenum in the herbage of certain pastures. In America molybdenum poisoning has been reported from California. The report indicates that "molybdenum is an essential micronutrient of plants, but when certain plants are grown in soils containing 1.5 to 3.0 parts per million they store up as much as 20 parts per million, which is toxic to cattle."

"Weighting" horns of cattle to make them curve downward is the device of the exhibitor so as to make the animal conform in this respect to a definite standard set as a breed characteristic. In young animals eight-ounce "weights" are used for each horn, and for mature animals one pound each. The weights are usually left in position for a week and then removed for several days; the process is repeated at regular intervals. Good judgment must be exercised in applying weights, and even then there sometimes develops evidence of pain at the base of the horn. If ignored a fracture may result. That's where the veterinarian is called in and finds a badly deformed horn and possible frontal sinus involvement.

Because of a shorter life span in most animals, potential fluorine poisoning does not have the same significance that it has in man. Animals may get fluorine from raw rock phosphate in mineral mixtures, which have fluorine as a constituent; in fluorinated drinking water, and to an extent on vegetation. Fluorosis in animals, as in man, is first noticeable because of the mottling of the teeth, but this does not affect digestion. However, when there is more than 3.5 to 4% of fluorine in acid phosphate and dicalcium phosphate used in mineral mixtures for cattle and swine over a prolonged period some softening of the teeth has been observed. In at least one state the sale of phosphorus supplements containing more than 0.2% of fluorine is prohibited. Bone distortion observed in chronic, endemic fluorosis in man has not been recorded in animals.

"Mule's operation" is the device of Mr. J. H. W. Mules, an Australian sheep breeder. It consists in removing folds of skin from the crutch of ewes. These folds if not removed become urine saturated and then attract blowflies. The work was originally done by means of the Burdizzo pincers, but now an instrument known as the "rolcut secateur" is used, with it the full length of the blade cuts at once. Also if the tip of the vulva is misdirected, usually towards the left, a small elliptical piece of skin is removed from the convex side of the vulva. Contraction of the cicatricial tissue when it heals straightens the organ.

Diethylstilbestrol in a dose not exceeding 15 milligrams, not to be repeated, implanted subcutaneously — usually in pellet form — in young tom turkeys, when they reach the fighting and strutting stage—generally at about 20 weeks of age—is said to subdue them to a degree that they do not molest one another. This synthetic hormone does not seem to fatten turkeys as it does cockerels. The effects of the diethylstilbestrol begin to gradually wear off about six weeks after implantation. There is no evidence to indicate that diethylstilbestrol is carried over in their flesh—an item of interest to prospective consumers.

Some day when you are asked to estimate the weight of a bovine, get out your tape measure and try the device of Extension-man Johnson of the University of Minnesota. His system is to first get the animal's heart girth in inches taken just back of the fore limbs. Second obtain the length in inches of the body extending from the tuberosity on the acromian spine on the scapula to the pin-bone. Now square the heart girth, multiply the result by length of body, and divide this total by 300 and you have the weight. The error varies from 3.6 to 5%. As an example, if the heart girth is 80 inches, this squared is 6400. If the 6400 is multiplied by a length of 60 inches the result will be 384,000. Dividing the 384,000 by the arbitrary figure 300 there is obtained 1280 which is the estimated weight in pounds of the animal.

Veterinary Problems in Poultry Production

By W. R. HINSHAW, D.V.M., Ph.D., Davis, California

Research work at the Wisconsin experiment station by Rosenberg, McGibbon and Herrick (Proc. Eighth World's Poultry Congress, Copenhagen, 1948:745) lend support to the hypothesis that resistance and susceptibility to cecal coccidiosis are inherited. Additional investigation will be necessary to determine whether or not a high degree of resistance can be established. Following one generation of selection a difference of survivability of 35.3% was observed between offspring of two lines of chickens, one a susceptible and the other a resistant strain.

At intervals during the past year, the writer has emphasized the need for veterinarians to urge their poultrymen clients of the need for caution in selecting sources of chicks or eggs for their flock replacements in order to avoid purchasing pullorum disease with their replacements. It is the time of year again when this caution should be given. Only by purchasing replacements from hatcheries that are in a position to furnish stock from pullorum-free flocks, can poultrymen be sure that they will not lose chicks from this disease. U. S. Pullorum Passed and U. S. Pullorum Clean grades are the two grades which will insure freedom from this disease.

The proceedings of the Eighth World's Poultry Congress held in Copenhagen, Denmark, in August, 1948, have just been received. A total of 132 papers were presented at the scientific section of the Congress. Of these, 33 (25%) were on some phase of disease or disease control. Most of these papers were in the nature of reviews on recent developments on the common diseases, but there were three papers presented on a new one. It is called "round heart disease" because of the change in contour of the heart, and according to one of the reporters (D. Luke) symptoms are usually absent; the birds being found dead or being seen to fall over in the runs.

Round heart disease is prevalent in Ireland; in Germany, where it is known as "egg heart"; in New Zealand, where it is called "enzootic fatal syncope"; in Scotland, and in Denmark. The etiology is unknown. Theories include infection, toxic substances and hereditary defects.

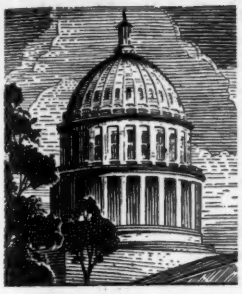
Post-mortem findings reveal dilatation and hypertrophy of the heart, and a yellowish discoloration of its muscles as a result of fatty infiltrations. No treatment has been satisfactory.

One of the important causes of abnormal white barring of feathers in Bronze turkeys is a deficiency of the amino acid lysine (Fritz *et al. J. Nut.* 31:3887,1946). The turkey poult's requirement for this amino acid is 1.3 times that of the chick. Good sources of lysine include fish meal, meat scrap, dried skim milk, soybean oilmeal, and dried brewers' yeast.

The scientific names of the eight valid species of coccidia known to occur naturally in chickens are as follows: *Eimeria acervulina, E. brunetti, E. hagani, E. mitis, E. necatrix, E. praecox, E. maxima,* and *E. tenella.* In turkeys, only two species have been described, *E. meleagridis* and *E. meleagrimitis.* Neither of the turkey species are infective for the chicken and likewise the chicken species have not been proved pathogenic for turkeys.

An important factor to be considered in recommending the use of any drug is its effect on feed consumption. To determine this for sulfaquinoxaline, Singsen, Scott and Matterson (*Poultry Science* 27:627, 1948) incorporated the drug at various therapeutic levels in the feed of chicks up to eight weeks of age. They found that 0.01 or 0.02% did not reduce the growth of the chicks, 0.03% definitely retarded their growth. Feather development was not affected by sulfaquinoxaline at any levels fed. They further reported that the drug has a tendency to increase slightly the efficiency of feed utilization.

In the experiment referred to in the foregoing, the authors showed that sulfaquinoxaline could be fed continuously in feed of young chicks at a 0.02% level without affecting their growth. In another paper Grumbles, Delaplane, and Higgins (*Poultry Science* 27:605, 1948) show that about half this percentage of the drug, fed continuously to market age, caused a marked reduction in mortality from cecal and intestinal coccidiosis, as compared to untreated controls. They used 0.0125% (¼ pound per ton) of the drug for nearly 20,000 chickens with an average mortality of 1%. The average mortality in 3000 controls was 17.4 per cent. Both *E. tenella* and *E. necatrix* infection existed simultaneously in the outbreaks.



Your Income Tax Return For 1948

By HAROLD J. ASHE
Tax Counselor
Compton, California

THIS year the taxpayer, in making out his individual return, may be faced with a new tax problem. If he is married he must decide whether to file a joint return with his wife, or whether tax savings can be made by filing separate returns.

For many years taxpayers have clamored for equality with taxpayers in community property states in splitting income for tax purposes. However, there are situations in which income splitting in a joint return may increase the total tax.

Here are the distinguishing characteristics of separate and joint returns of husband and wife:

Joint Returns:—Combine all income of both husband and wife, constitute a return for both. Form 1040 now provides for income splitting. Income is split in two parts. The tax is computed on one-half the taxable income, then multiplied by two to get the combined tax. Result is a lower rate than if income were not split. On a joint return of, say, $10,000 (after deductions and exemptions) tax woud be computed on $5,000, multiplied by two. Highest tax rate would be 26% on top $1,000. If taxes are not split, the top bracket will be 34%.

Separate Returns:—Show income only of each spouse. Example: husband has $12,000 income (after exemptions and deductions). His wife has $2,000. The husband would have $2,000 in the 38% bracket.

Who May File Joint Returns

Only husbands and wives may file joint returns. It is not required that they live together to use split-income provisions, provided they are not legally separated or divorced. Marital status is determined by the status on December 31, 1948. If married on the last day of the year they are deemed married the entire year for tax purposes. If legally separated or divorced on December 31, 1948, they are considered single for the tax year.

If one spouse died during 1948, a joint return may, nevertheless, be made, reporting both the survivor's income, if any, and the income, if any, of the deceased spouse, and dividing the combined income in a joint return.

Exceptions

A Joint return may not be filed if either spouse was a non-resident alien at any time during 1948. Neither can a joint return be filed if the couple maintain different taxable years.

Different Accounting Methods May Be Used by Husband and Wife

Even though husband and wife use different accounting methods they may file joint returns. For example, the husband may be on an accrual basis, the wife on a cash basis. The Internal Revenue Bureau emphasizes that both methods must correctly reflect the true income of each spouse.

Each Responsible for Entire Amount of Unpaid Tax

Where joint returns are filed, taxpayers must both sign and each is responsible for the entire amount of the unpaid tax, or any tax subsequently levied against the return. In event of a refund, the treasury check will be payable to both spouses, and both must endorse the check to cash it.

Dependents

When husband and wife file separate returns they not only must report their own income, but must take only their own dependents as exemptions. The spouse furnishing principal support of a dependent must take that dependent as an exemption even though it may be to the tax advantage of the other spouse to claim that dependent. Dependents claimed by each taxpayer must, by relationship, come within the legal definition of dependent.

However, in a joint return it is not necessary to separate dependents between husband and wife. The test of whether the dependent is legally deductible is determined by the relationship to either spouse.

When Tax Savings Are Made by Joint Returns

The joint return, if combined income after exemptions and deductions is over $2,000, will effect tax savings if:

1. One spouse's income is substantially greater than the income of the other spouse;
2. One spouse has income; the other sustains a loss;
3. The entire income of husband and wife is earned by one spouse.

Savings Through Separate Returns

Frequently separate returns are advisable when medical expenses have been excessive for one spouse, and each has separate income. Medical expense deductions are limited to the amount in excess of 5% of adjusted gross income. On a joint return the couple is considered a single entity.

Example: Each spouse has $5,000 income, or combined income of $10,000. Superficially, it would appear immaterial whether each filed separately or combined and split income for the same apparent tax result. In fact, there is considerable difference in the choice made. The wife has $1,000 medical expenses, the husband none. On a joint return $500 of this medical expense (5% of $10,000) is non-deductible, leaving only $500 deductible from the joint return. In a separate return, the wife would deduct $750 (5% of $5,000, or $250 being non-deductible). On medical deductions, alone, a joint return would be reduced to only $9,500. By separate returns total income would be reduced to $9,250 ($5,000 for the husband plus $4,250 for the wife). Assuming each have substantial other personal deductions, the above example would result in a material tax saving if separate returns are made.

Separate returns may be advisable where both husband and wife have sustained capital net losses, if both have substantially the same income. Tax regulations limit the amount of a capital loss which can be deducted from ordinary income to $1,000. In a joint return only $1,000 may be deducted. In separate returns each may take up to the $1,000 maximum. This advantage may be illusory if one spouse has large income, the other a nominal income. This is because the larger income of one spouse would place his un-split income in much higher tax brackets, thus offsetting the tax advantage of the extra $1,000 of deductions accruing to the other spouse in a much lower tax bracket.

This situation, as well as others, points up the wisdom of figuring the income tax, when in any doubt, both separately and jointly.

Saboteurs of the Public Health

In nearly all parts of the United States, vital research and teaching in health problems are being delayed by antivivisectionists, while anxious disease victims await the drugs, the knowledge, and the skill to save them. These tragic delays are brought about by antiscience cultists who play upon public ignorance and public indifference. Working like the fanatics they are, they defeat the will of an apathetic public.

This waste of life, this attack upon mankind must be stopped, and the only possible way to do it is to arouse the public to the vital relationship between medical research and education, and their own health and welfare.—A. C. Ivy, M.D., Ph.D. *Secretary, National Society for Medical Research.*

Ancient Medical Doctrine Rises from Its Ashes

The humoral conception of morbidity expounded by physicians from Hippocrates to Harvey, and beyond, can no longer amuse the jeer-minded critics of ancient medicine for, when the chemistry of the humors was deserted for the histopathology of the flesh, the joke was on science, because it willfully wandered away from the so-called blood picture and its relations to metabolism, or life itself. At least, the ancients charted their course toward blood volume, blood pressure, blood minerals, blood proteins, blood ferments, blood types, blood sedimentation, blood-cell concentration, hemoglobin, hemasiderin, hemapoiesis, antigens, the anemia complex, Rh, and other factors associated with humors.

Briefly told, modern medicine has come back to the quirks of the humors recognized by the pre-Christian doctors as *the* thing to reckon with in the study of disease.

The 1906 edition of *Keen's Surgery*, a comprehensive treatise in two volumes, opens its chapter on "Examination of the Blood" with: "Hematology, if *sanely* interpreted, is of *real* value in surgical practice." (Italics ours) There is but to compare what this sentence reveals, as of the turn of the century, with the pages on hematology now required to round out the knowledge of humors in medical practice.

In the historical sense, discovering the circulation of the blood (Harvey, 1628) swayed investigational medicine away from rather than toward the body liquids, for, it was not until the dawn of the twentieth century that hematology became a leading chapter of medical science.

The Two-Gram Daily Dose of **Phenothiazine** for **Strongylosis** of the Horse*

By W. W. DIMOCK†, D.V.M.
Lexington, Kentucky

SMALL nematode parasites of the large intestines of horses, particularly *Strongylus vulgaris*, one of the three species of strongyles, have long been known for their serious and extensive parasitic action. Limitations of regular periodic treatment and difficulty of prevention under usual farm-management procedures have been equally well known.

The high efficacy of phenothiazine in full therapeutic dose, one ounce, against the small nematode parasites that live and reproduce in the large intestine of the horse being well established, tests were started to determine the effectiveness and practicability of a small daily dose. The amount arbitrarily decided upon for the test was two grams. As the tests were to be conducted on breeding farms, in the Bluegrass Area of Kentucky, and all the animals were valuable, it was essential to proceed cautiously. It was necessary to establish criteria as to length of time required to feed a two-gram daily dose to establish anthelmintic action. Would an ounce given over a 15-day period be less effective than, equal to, or more effective than an ounce at one dose? Likewise the tolerance of a horse for a small daily dose of phenothiazine given over a period of days, weeks, and months had to be determined.

Trials so far conducted in this project on the effectiveness of a small daily dose of phenothiazine alone, and as a supplement to preventive measures and treatment at intervals, for strongylosis of the equine species have given promising results. Two grams daily for 14 days stops egg production by strongyles and other small nematodes, common to the large intestine. It renders many of the eggs passed infertile, it kills the worm itself, and possibly is effective against the infective larval forms of strongyles before they can begin their migrations through the tissues of the host.

At the start, the two-gram daily dose was continued for 15 days, during which time the horse, of course, received one ounce of phenothiazine powder. For the following seven to 10 days no phenothiazine was given. The feeding of two grams daily demonstrated in all cases, that in a horse having a high strongyle egg count at the beginning (flotation method), the egg count would drop to zero, or to an exceedingly low count, at the end of 15 days. As to tolerance, there was no evidence of untoward reaction to the drug, so the feeding period was extended to 21 days, withheld for 10 days and repeated. For convenience and regularity of dosage, a good plan is to start on the first day of the month, feed for 21 days, discontinue for the remainder of the month, and then repeat. In some cases horses have been continued on a two-gram daily dose for 30 to 60 days without the slightest evidence of ill effect.

With slight variations during a three-year period, this method of administering phenothiazine in the feed for 21 days, discontinuing it for 10 days, and then repeating, has been carried out on more than 500 animals in the field, and in no single instance has there been manifest clinical evidence of injury from the chemical; in fact, there has been evidence of improvement in the animals treated. In some few cases where there was marked evidence of unthriftiness and a decided sub-normal condition at the end of two to four weeks there has been a striking, and even spectacular, improvement in the general physical condition of the animal. To date, the tolerance of horses for a two-gram daily dose of phenothiazine, continued without interruption for weeks and months cannot be said to be fully established, although in a few instances we have fed it for periods up to 60 days without producing any evidence of injury to the animal. Further tests will show what ill effects, if any, may come from a long-time feeding of the two-gram daily dose. Observations and such tests as can be made also will determine how effective the treatment would prove in ridding horses, and eventually pastures, of the small nematodes so numerous and common in the cecum and colon of horses. There

† Department of Animal Pathology, Kentucky Agricultural Experiment Station, Lexington, Kentucky.
* The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director.

also remains to be determined what effect, if any, this method of administration will have against infective larvae upon intake and before they have started on their usual migrations.

One step in the life history of the *S. vulgaris*, not yet determined with finality, has a direct bearing on the value and necessity of preventive measures, and also, to a large degree, on how completely effective a small daily dose of phenothiazine will prove, where horses are continuously exposed to infective larvae. If infective larvae, and particularly larvae of the *S. vulgaris*, can penetrate the mucous membrane of the digestive tract between the mouth and the ileocecal valve, a small dose of phenothiazine, once daily, could be expected to destroy few if any before they start their migrations. On the other hand, if infective larvae, in part or all, pass on to the cecum and colon before starting their migrations, then the small daily dose would be expected to destroy at least some of them before they could bore into the wall of the intestine beyond the reach of the anthelmintic. Since the small daily dose for 15 to 30 days will stop egg production and apparently kill adults, it may be assumed that it will kill larval forms if brought in contact with them. This is of special importance in the case of young foals from the time they start to graze until weaning time, a period during which anthelmintic treatment is (1) difficult to administer, (2) intake of infective larvae is usually high, (3) there is maximum vulnerability on the part of the host, and (4) when the damage is often extensive, and in certain structures largely irreparable.

The *S. vulgaris* has been observed in the anterior mesenteric artery of a foal 30 days of age and has been reported as having been found in even younger foals. From six months of age to maturity is also a period of great susceptibility and often of heavy exposure, but it is easier to treat animals of this age by a single therapeutic dosage or by adding the anthelmintic to the feed than to treat unweaned foals. In colts six months to two years of age that die of parasitic colitis, large active verminous aneurysms in the mesenteric arteries, and abscesses along the bowel wall, as well as extensive damage to the mucosa of the cecum and colon, are usually found on necropsy. It is evident and has long been known, that the arterial and other lesions had their beginning weeks and months before the death of the animal.

There is considerable evidence that phenothiazine, in large or small doses, renders many parasite eggs nonviable, therefore the continued use of this method of administering phenothiazine has great possibilities if properly and consistently used, and will, in time, contribute much toward the eradication of the strongyle parasites from a farm and groups of horses. There will always be many sources of reinfestation on the farm and elsewhere; perhaps the two most common sources which are difficult to deal with are new animals brought onto a farm, and horses permitted to graze, even temporarily on grass areas where contamination with parasite eggs is heavy.

The Phenothiazine Mix

To administer a small, measured amount of an anthelmintic to livestock daily over a period of weeks, the procedure should be simplified to a point of convenience and economy, at the same time maintaining regularity and reasonable accuracy in dosage. The two-gram daily dose, mixed with a palatable vehicle and added to the feed, meets all these requirements and eliminates many of the objectionable features of giving a full therapeutic dose. The procedure followed has been to use as a vehicle two or three dry and granular feed and mineral substances with which phenothiazine will blend readily. For this purpose distiller's dried solubles and steamed bonemeal, mixed equal parts by weight, are satisfactory. The measured amount of the mixture selected to carry the two grams of phenothiazine was a heaping teaspoonful. A later vehicle used was compounded in the proportions: steamed bonemeal, one part; distiller's dried solubles, two parts; and linseed oilmeal, one part by weight. When 120 grams (4 ounces) of phenothiazine is mixed with one pound of the above, there will be approximately 60 heaping teaspoonfuls in the final mix. The phenothiazine should be sifted to break up all small lumps and make sure of uniform distribution. Distiller's dried solubles on the market differ somewhat chemically and also in physical characteristics. The fine granular form makes a better mix than the small flakes. Not all steamed bonemeal is equally fine; coarse particles and hair, if present, should be sifted out. Ingredients may be sifted separately or the final mix may be sifted. What is best and advisable depends upon the physical properties of the material used as a vehicle. If the final mixture is firmly packed in the container, it should be stirred before measuring out the heaping teaspoonful. This will assure greater uniformity in the measured dose.

The nature of the vehicle or diluent is important only in that the phenothiazine mixes well with it, and the final mixture is dry and

granular, so that a heaping teaspoonful or other measure used, gives a fairly regular amount of the anthelmintic. The mixture can be made up so as to be exact for any selected measuring cup. It is important only that the portion of the mix which is given contains two grams of phenothiazine.

For those parasites of the digestive tract for which phenothiazine is effective, the standard therapeutic dose (30gm) is correct, and if the animal is not subnormal nutritionally or otherwise sick, the result will be satisfactory. Horses that have been on a low protein ration, or are subnormal physically, or have only a subsistence level of vitamin A, or are allergic to phenothiazine, sometimes, after receiving a full therapeutic dose of phenothiazine, suffer a mild or severe systemic reaction and on occasion die. This explains why horses on pasture, or that otherwise are receiving an adequate, balanced diet, have a high tolerance for phenothiazine, while those stable fed, on dry feed low in protein and deficient in vitamin A, have a low tolerance and may suffer visible reaction from a standard therapeutic dose. Based on observation and experience, the opinion has been expressed that some horses, fed hay and grain and good pasture, may manifest susceptibility to even a divided dose of phenothiazine. The unfavorable reaction in such cases apparently has no relation to nutritional factors.

For a horse carrying a heavy parasitic load of strongyles, a full therapeutic dose of phenothiazine may be preferred to the small daily dose. If, however, the animal's physical and nutritional condition is not up to normal it is advisable to administer the anthelmintic in divided doses.

Early reports on the use of phenothiazine as a treatment for intestinal strongylosis of the equine species were to the effect that horses were highly resistant to a large dose of phenothiazine. Further experience has demonstrated that some may die from a standard therapeutic dose, 30 grams for an 800 to 1200 pound horse, that some manifest a mild to serious systemic reaction, and some show no clinical symptoms. Thus it may be assumed that in the non-clinical cases some systemic, kidney and blood changes may take place, but are of a temporary nature. The following cases came under observation.

Four stallions, that had been stable fed for several years, were each given a one ounce dose of phenothiazine powder. One showed a mild reaction and another a more severe reaction. Both experienced hemoglobinuria. The other two stallions, same breed and about the same weight, treated at the same time, manifested no clinical evidence of a reaction. In another instance a two-year-old colt, that had been stabled most of the time for over a year, and fed hay and oats exclusively, was given one ounce of phenothiazine in the feed. He suffered a severe systemic reaction, passed quantities of clotted blood in the urine, and later died.

Measuring the Results

The use of a small daily dose (2 gm) of phenothiazine in the feed over a period of three years has been shown to be most effective in reducing the number of strongyle eggs in the feces in a large number of horses. The tests and observations, with two exceptions, were made on horses kept on the farm under natural procedures of stabling, feeding, and pasture. On five farms, where the number of horses varied from five to 100 or more, the animals received the small daily dose and the results in all instances were entirely satisfactory. Anthelmintic action was high with no evidence of ill effect on the host animal, the incidence of those types of sickness definitely related to parasitisms has been low and the health of the horses has been above the average. From diverse angles of analysis the results have come up to every requisite of expectation.

Not all horses were given a check examination of feces before and after treatment. In both large and small groups spot testing was carried out to obtain an approximate estimate of the parasitic load as measured by the number of eggs observed on examination. (Flotation method.)

Table I gives the results of reduction in parasite eggs for four horses fed and kept under their regular environment on the farm.

Table I

	Ascarids	Strongyles			
Horses No.	1 2 3 4	1	2	3	4
July 21	0 0 0 0	++	+++	+++	+++
August 2	0 0 0 0	3 Eggs	0	1 Egg	2 Eggs
August 22	0 0 0 0	0	0	0	0

Table II gives the results of egg reduction and autopsy findings on two colts confined in site eggs and autopsy findings are given below.

Table II

Weanlings	Ascarids		Strongyles	
A and B	A	B	A	B
December 2.....	0	+	++	+
December 14.....	0	+	0	0
December 23.....	0	+++	0	0
December 29.....	0	+++	0	0

In the fall of 1947 two weanlings became

available for test feeding of phenothiazine and autopsy at the close of the test. The daily dose of phenothiazine was two grams in a heaping teaspoonful of the standard mix added to a grain feed of crushed corn and oats and continued for 28 days, December 2nd to December 29, inclusive. Both weanlings had been treated with carbon disulphide and phenothiazine in September. Weanling A was affected with incoordination (wobbles). Weanling B had a form of chronic non-suppurative arthritis and contracted tendons in both front legs. Results of examination of feces for parasite eggs and autopsy findings are given below.

On the basis of egg count, these two weanlings were only moderately infested with strongyles at the start of the test. However, the results reveal that a daily dose of two grams of phenothiazine effectively stopped strongyle egg production and killed and eliminated the small nematode parasites of the large intestine, but had little or no effect on ascarids.

The two weanlings were sacrified at the end of the 28th day of the feeding test. One *S. vulgaris* was found in the cecum of weanling A and two in weanling B. These were attached to the mucosa of the cecum, were only about one-third normal size, and had not reached the egg production stage of development. No other parasites of the strongyles and related species were found. Each had a verminous aneurysm of moderate size in the mesenteric arteries. There were no other parasitic lesions and no gross lesions in the mucosa of the cecum and colon.

The two-gram daily dose of phenothiazine for 15 to 30 days when given to adult horses, kept on the farm under daily exposure, has given almost exactly the same results as in the case of young animals.

There is evidence, accumulated from careful examination post mortem, that adult horses heavily parasitized when young and having extensive chronic lesions, may later suffer from a heavy new invasion of infective larvae which, in turn, set up acute reactions and cause further tissue injury, but this is believed to be exceptional. On the other hand, a few aged horses have exhibited at autopsy, only a light intestinal infestation and a small, healed, verminous aneurysm in which no worms were discovered. Age resistance to certain kinds of parasites is accepted, and it is apparent that some animals develop a marked degree of tolerance to many species of parasites. It is also known that the young suffer more severely than the aged from most kinds of parasitisms. In horses there is a high degree of age resistance to ascarids. For strongyles, the degree of age resistance is not so complete, certainly not as regards intestinal invasion. There is a wide acceptance of the belief that some internal parasites produce a reaction in the host animal comparable to immunity. On this basis it would be desirable for a young animal to harbor parasites in numbers just short of sufficient to cause a serious systemic reaction and tissue damage beyond what would in time be repaired.

Aged horses, on the whole, may be considered wholly resistant to tissue and blood invasion of infective strongyle larvae or in case of invasion the parasite encounters an environment sufficiently unfavorable to prevent development and result in its death. Regardless of whether infective strongyle larvae do or do not invade the tissues of mature horses, or can or cannot survive following initial invasion, a mature horse may harbor in the large intestines thousands of strongyles and related, small nematodes, and are in most instances, active spreaders of viable eggs. Thus there is ample evidence that both young and mature horses should be kept as free from parasites of the digestive tract as measures now known make possible; especially should they be protected against *S. vulgaris*.

Every known measure of prevention should be interwoven with accepted practices of care and management of horses. This would require modification of only a few established procedures and practices, long followed in farm and horse management. If preventive measures are instituted to reduce the perpetuation, the source, and the intake of infective strongyle larvae and these measures are supported by the regular and systematic feeding of the two-gram daily dose of phenothiazine, the results will be noticeable shortly. New additions to a farm, on which a parasitic reduction program is well under way, should be treated with a full therapeutic dose of phenothiazine or held in stable two weeks on the two-gram daily dose before being turned into pastures and/or paddocks used by other horses.

The more regular and frequent use of full therapeutic doses of anthelmintics may well be a major part of a parasite prevention program, especially at the start. Finally, from the tests so far conducted and results obtained, the two-gram daily dose of phenothiazine for strongyles and related species will, if administered with regularity, materially add to the effectiveness of other measures now in use. Further, there is a possibility that it may be sufficient, as a single measure of procedure, to free horses of strongyles and keep them free from birth to the time when they are no longer needed or useful for the purpose for which they were primarily produced.

Preliminary Report on Use of Sulfamethazine & Penicillin in Bovine Mastitis

By R. W. FULLER, D.V.M.*
Batavia, New York

BOVINE mastitis is recognized as the most costly animal disease of the dairy industry. Some of the obvious losses are the permanent damage to the udder, reduced milk production, production of abnormal, unsalable milk, and the forced elimination from the herd of heavily infected, unprofitable dairy cows for beef. *Streptococcus agalactiae* has been reported[1] to be most commonly associated with bovine mastitis, with other streptococci, staphylococci and coliform organisms of much less importance. However, it is felt that there is not necessarily a direct correlation between the bacteriological laboratory findings and the incidence of clinical mastitis. The present paper is a progress report on the treatment of clinical mastitis with sodium sufamethazine as diluent for penicillin, administered by intramammary infusion, in New York State institution herds, carried out under the State Mastitis Control Program.

Much progress has been made in recent years in the treatment of bovine mastitis through the use of therapeutic agents. Favorable results have been reported frequently following the administration of penicillin or sulfonamides[2, 3, 4, 5, 6]. Bryan and his associates[2] reported favorably on treatment with penicillin both parenterally and by infusion, with simultaneous oral administration of sulfanilamide. For some time, a combination of sulfanilamide in oil or sodium sulfamethazine, and penicillin has been used by Johnson and Roberts[7]. The conclusion reached by these workers is that " . . . . a combination of sulfonamide and penicillin produced better bacteriological and clinical results with fewer injections than either one of the drugs administered separately." Slanetz and Allen[8] have reported highly satisfactory results with the combination of penicillin and soduim sulfamethazine. In their work, immediate improvement in the condition of the cows was noted, with no signs of toxicity and no marked reduction in milk production in any of the animals treated.

The success of any mastitis control program depends to a great degree on the management and sanitary measures used to prevent injury and infection[1,5]. Therapeutic agents can reduce immediate infection, but they do not prevent reinfection[5]. *The objective of a control program is to return the affected cow to the milking line at the earliest possible moment by good treatment procedures, and to keep her there by sound herd management and sanitary practices.*

Avoidance of teat and udder injury is of primary importance in the control of mastitis [9,10]. Several inexpensive changes in barn and stall construction were found to be most important. Among these was adjusting the stall to the size of the animal, providing adequate length and width. Sufficient dry bedding was provided to protect the udder against chilling. Sharp inclines at entrances, high door sills or other obstructions on which teats and udders might be injured, and inadequate passageways, were removed or corrected. In one herd having a high percentage of mastitis, where cows were milked three times daily, it was found that each animal was walking an average of 9 miles a day to and from pasture. Udder exposure to injury from stumps, brambles, and the mechanics of walking was excessive, and those having large pendulous udders were subject to maximum exposure. When this situation was corrected, the percentage of mastitis was markedly reduced. As there was no other change in herd management, it is suggested that udder injury may have been the primary cause.

History

Approximately 127 cows in various state institution herds have been treated with sulfamethazine and penicillin. In all herds, management and sanitation could be considered about average. In most instances a full-time herdsman was employed, but in others ideal supervision was not attained. The majority of the animals were tended and milked by inmates of the institutions. Many of these workers are mentally irresponsible and consequently are not readily adaptable to a new routine. Segregation of infected cows or placement of infected cows at the end

*Veterinarian, State Institution Farms Bureau.

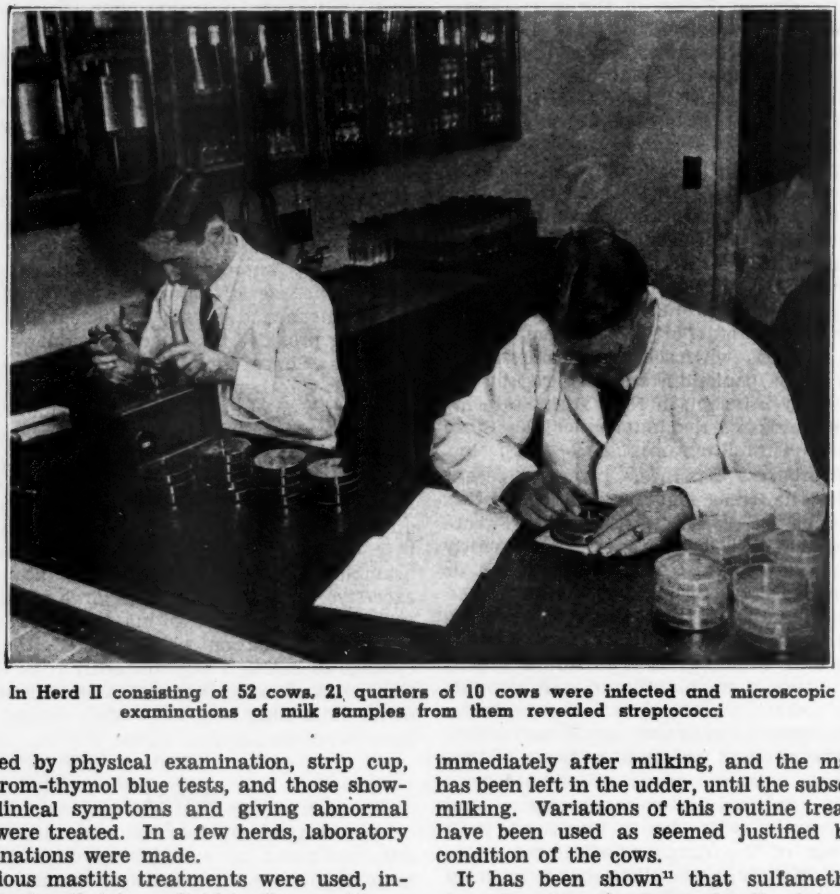
of the milking line was not generally practiced.

Some barns had badly worn floors and the liquids held there were an ever present source of infection. Narrow doorways and smooth slippery floors caused crowding and occasional injuries. Marshy pastures and muddy lanes were additional hazards in rainy seasons. Due to war-time shortages, teat cup inflations were frequently rough and a source of irritation.

These herds had been under treatment for mastitis for several years. Cows were judged infected by physical examination, strip cup, and brom-thymol blue tests, and those showing clinical symptoms and giving abnormal milk were treated. In a few herds, laboratory examinations were made.

Various mastitis treatments were used, including tyrothricin, colloidal silver oxide, a sulfanilamide-sulfathiazole-urea combination, and sulfanilamide in oil. Improvements in management and sanitation were also instituted. Very gradual improvement in the condition of the cows was noted, but the treatments were continued over long periods with heavy losses in milk. It was usual for animals to be out of production for 10 days to two weeks, when these treatments were used.

The sulfamethazine-penicillin treatment program was instituted in July 1946, and herd surveys have been made at varying periods. Early in the program, the routine treatment consisted of 50cc. sterile solution sodium sulfamethazine 10% weight/volume, plus 25,000 or 50,000 units of penicillin, infused into each quarter. Since then, 50cc sodium sulfamethazine 25% w/v, plus 50,000 units of penicillin has been used in the same manner. Either four infusions at 12-hour intervals, or two infusions at 24-hour intervals, have been given immediately after milking, and the material has been left in the udder, until the subsequent milking. Variations of this routine treatment have been used as seemed justified by the condition of the cows.



In Herd II consisting of 52 cows, 21 quarters of 10 cows were infected and microscopic examinations of milk samples from them revealed streptococci

It has been shown[11] that sulfamethazine, given in adequate dosage by the intramammary route will establish effective blood concentrations and, in turn, effective tissue concentrations in the secreting udder. It is obvious that adequate concentrations of sulfonamides are important for good therapy. Using the 25% w/v material in adequate volume by under infusion establishes effective blood levels and, in turn, effective tissue concentrations in the udder. On this basis,

adequate dosage should be put into the udder. To avoid great volume in udder infusion, the concentrated drug can be used.

Clinical Cases

The treatment in two representative herds will be described in detail. In herd I, totaling 89, a group of 19 cows, with 34 clinically affected quarters, was treated in May 1947. A second group of 13 additional cows in this same herd were found infected and treated late in October. In herd II, 10 cows of a total of 52, with 21 infected quarters, were treated in May 1947. Treatment was instituted on the basis of physical examination, strip cup, and survey 10 days later, 25 of the 28 infected quarters, or 90%, were clinically free of mastitis.

Herd II.—Microscopic examination of incubated quarter samples and cultural tests showed streptococci in all 21 infected quarters, and in six additional quarters still producing apparently normal milk. Dosage in all cases in this herd was 50cc sodium sulfamethazine 10% w/v with 50,000 units of penicillin per quarter. Four infusions at 12-hour intervals were administered to four cows. Examination of quarter samples six weeks after treatment showed clinical and bacteriological recovery of all treated quarters. Five months after

TABLE I.—TREATMENT OF CLINICAL MASTITIS WITH SODIUM SULFAMETHAZINE AND PENICILLIN.

Herd	Group	Units Penic. per quarter	Amt. of Na. Sulfamethazine per quarter	No. cows	No. quarters clinically affected†	No. treatments required for recovery			Clinical recoveries	No. quarters not recovered
						2	4	6		
I	1	25,000	5gm*	19	34		32		94%	2
	2	50,000	1st day—12.5gm** 2nd day—5gm*	13	28		25		90%	3
II		50,000	5gm*	10	21	11	8	2	100%	—

* 50cc. 10% w/v sodium sulfamethazine.
** 50cc. 25% w/v sodium sulfamethazine.
† All milk from these quarters was unsuitable for human consumption at start of treatment.

bromthymol blue tests. In herd II, bacteriological examinations were made as an additional check. All quarters treated were producing clots, pus, flakes, watery, and/or off-colored milk which was discarded as unsuitable for human consumption.

Herd I, Group 1.—The dosage used was 25,000 units of penicillin plus 50cc sodium sulfamethazine 10% w/v, per quarter, infused four times at 12-hour intervals. Clinical recovery occurred within one or two days in all but two quarters. On examination 10 days after treatment, 32 of the 34 infected quarters, or 94%, were clinically recovered. Approximately two months following treatment, five cows were disposed of as low producers. Five months later, the herd survey showed reinfection in six quarters of two cows of the 14 remaining. These two cows had a long history of chronic mastitis, and were used for meat on the premises. All others were clinically free of mastitis.

Group 2.—The dosage used in this group was 50,000 units of penicillin dissolved in 50cc sodium sulfamethazine 25% w/v on the first day, and in 10% w/v on the second day, per quarter. Two infusions were administered each day at 12-hour intervals. At the herd treatment, these four cows still were clinically free of mastitis. Although streptococci were again isolated from the milk of one of the quarters, all were producing salable milk.

Two infusions were administered at 24-hour intervals to six cows. Four of these were clinically and bacteriologically free of mastitis at six weeks, and, at five months, by microscopic examination, reinfection had taken place in one quarter. However, here too, the milk obtained from all quarters treated was vendible.

One cow, with two quarters clinically affected, and three quarters from which streptococci were isolated on the original examination, had four quarters both clinically and bacteriologically infected at six weeks. She was retreated with four infusions at 12-hour intervals. At five months, all four quarters were clinically normal and bacteriologically free of mastitis.

Streptococci were isolated from all four quarters of one cow, although clots were obtained from only one quarter. She was not examined at six weeks, but, at five months, although streptococci were still present, she was clinically cured and producing salable milk from all quarters.

The treatments used, and the results of treatment of the cows in these herds are summarized in table I. When checked at varying periods, as indicated on the chart, 94.6% were clinically cured, and were producing salable milk.

Acute Mastitis

Excellent results have been obtained in the treatment of acute mastitis with sulfamethazine and penicillin. Typical of these, are the following three given in detail. The clinical picture was characteristic. The cows had high temperatures, ranging from 104° to 106° F., accompanied by loss of appetite; udders were hot, swollen, and painful, and there was a sharp drop in milk production. A single udder infusion was given. The dosage used was 50cc sodium sulfamethazine 25% w/v and 100,000 units of penicillin per quarter. Within 24 hours, there was a marked reduction in fever, appetites had improved, and the local swelling had nearly all disappeared. All cows were back in full milk production within 36 to 48 hours after treatment.

In correlated work, excellent results have been obtained in the treatment of acute toxic cases, with oral or parenteral dosage of sulfamethazine. Frequently, udders in this condition are so acutely swollen that an adequate dosage cannot be administered by infusion and is not well absorbed. However, it has been shown[11] that, when an effective blood level of the drug is established, the drug is carried to all of the affected parts by the blood stream, and comes through the udder tissues in bacteriostatic amounts. In general, this would seem to be a more effective treatment than local infusions.

Previous to this type of treatment, usualy 10 to 15 days elapsed before the animals were again giving salable milk. Not infrequently, there was a considerable destruction of udder tissue, and an occasional animal died as a direct result of acute mastitis. For best results, treatment should be started as early as acute mastitis is diagnosed.

Discussion

It does not seem possible to evaluate a mastitis herd control program fully or accurately, on the basis of bacteriological findings alone. Every effort should, of course, be made to have bacteriologically negative milk. This is the ultimate goal. Quarters harboring pathogenic organisms are a source of infection for other animals in the herd even though the infection in such quarters may be quiescent. It is possible to sterilize the udder of the cow by infusion with sulfonamides, penicillin, or a combination of both, and the combination has been shown to be more effective than either remedy used alone.

From the dairyman's point of view, the important factor is the amount of salable milk the herd produces and the freedom from being forced to sell cows for meat purposes because of udder failure. Surveys of commercial herds have indicated that 10 to 30% of the cows are forced out of the herd annually, chiefly because of mastitis. Since mastitis control programs were instituted in the herds here reported, the percentage of animals excluded from the herds because of mastitis has been reduced rapidly. Because of the lower turnover of mature cattle, fewer unproductive head of young stock have been needed and only the superior heifers have been retained, thereby more fully insuring the future herd efficiency. At the present time, fewer animals are needed to produce the same quantity of milk that was obtained previously, and a smaller number of calves and heifers have to be maintained for replacement purposes. This has appreciably reduced milk production costs.

The current favorable status of these herds is the result of several factors. Although management practices certainly are not ideal at present, many improvements have been made. Considerable attention has been given to adequate and proper nutrition of these herds, and, as a whole, the individuals are undoubtedly in better physical condition than in previous years. Barn construction has been improved to the extent of providing adequate space for each individual animal, and other improvements made, with the object of avoiding injury. Immediate care is given to chapped or sore teats, and small abrasions and lesions at the teat opening which form sites for bacterial growth which may infect the udder. Milking equipment is much better serviced, and teat cups are carefully checked.

Personnel has responded favorably to instruction on proper preparation of the cows, and the rapid method of milking. The methods of proper milking have been so frequently given that they hardly need repetition here. However, failure to use these methods is a contributing cause of mastitis.

Several factors must be taken into consideration in deciding whether or not to treat an animal for mastitis. A physical examination of the udder should certainly be made, the milking history should be considered, as well as the quality and quantity of milk at the time of examination. Supplementary evidence, as shown by laboratory examination, is helpful. Treatment, therefore, must be largely based upon experience and judgment rather than on one type of test alone. Local treatment, with a combination of penicillin

and sodium sulfamethazine, was found highly efficient in restoring animals to profitable production in most cases of clinical mastitis. These treatments[7,8], in a high percentage of cases, render the udder free of mastitis organisms, and usually restore the animal to the point of producing salable milk with little drop from previous production levels.

From our experience so far, we believe that treatment alone, even with the superior agents now available, can not be effective in the control of mastitis within a herd, unless, at the same time, good management, proper sanitation, and adequate nutrition are employed. It is equally true, however, that, even when these latter factors are applied, mastitis will occur, though less frequently, and therapeutic agents, properly used, can be an invaluable aid in reducing tissue damage, and in aiding in the production of the maximum quantity of salable milk. Further the value of early treatment as a contribution to good sanitation should not be over looked. It decreases enormously the amount of infective material contaminating the premises.

Our evidence so far indicates that mastitis can not be eliminated rapidly from a mature herd. Older animals, showing varying degrees of induration of the udder, frequently require local treatment one or more times during a lactation period to keep them in profitable production. If further injury to the udder can be avoided, and the injured tissue heals during the rest period, little further trouble may be experienced. The greatest factor, however, in controlling mastitis in a herd, lies in the proper handling of the replacement heifers, introduced into the herd, to avoid the basic causes of mastitis, and in the prompt and early treatment of such cases as do occur. The success of a mastitis control program is largely dependent, also, on the intelligent cooperation of the herdsman and those handling the cattle.

Few would contend that mastitis can be entirely eliminated from a herd with the procedures now known. Our experience, and the experience of others, definitely show that, with intelligent management and proper use of agents now available, mastitis need no longer be an economic burden in any dairy herd.

Summary

1. A total of 64 cows with clinical mastitis have been treated with sodium sufamethazine and penicillin since July 1946. These cows were in New York State institution herds, and have been treated under the State Mastitis Control Program.

2. The dosage used routinely was 50cc sodium sulfamethazine 10% w/v or 25% w/v, plus 50,000 units of penicillin per quarter, infused immediately after milking. Depending upon the severity of the infection, a single infusion, two infusions at a 24-hour interval, or four infusions at 12-hour intervals were given.

3. Treatment of 42 cows, with 83 clinically infected quarters, is given in detail.

4. Milk suitable for human consumption was regularly obtained in three to five days with the sulfamethazine-penicillin treatment, in comparison with 10 to 15 days, which was usual when other treatment was used.

5. When checked at varying periods, 94.6% of the affected quarters were clinically cured.

6. The sulfamethazine-penicillin treatment was equally effective in all cows; age and stage of lactation were unimportant.

7. Treatment of acute mastitis with sufamethazine and penicillin resulted in marked improvement in 24 hours, and cows were back in full milk production in 36 to 48 hours.

8. There were no signs of udder irritation from the therapeutic agent in any of the animals treated.

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Action of Hexachloroethane-Bentonite Suspension on the Rumen Fluke, Paramphistomum

By O. WILFORD OLSEN*
Angleton, Texas

IN studies on the anthelmintic properties of hexachloroethane-bentonite suspension, a medication developed by the writer for the removal of liver flukes from domestic ruminants, an opportunity arose for testing the treatment on an aged ewe and a milk cow that were infected with adult flukes of the genus *Paramphistomum*, trematode parasites of the digestive tract.

Parasites of the genus *Paramphistomum* and related genera of amphistomes, as will be shown elsewhere in this paper, have been reported as being injurious to cattle, sheep, and goats in other parts of the world. They are said to produce a clinical entity that is referred to in this paper as amphistomiasis. In this country amphistomes have not been extensively studied and comparatively little information is available, therefore, on their pathogenicity to our domestic ruminants. Considering the fact that these parasites have not been encountered often enough in the United States to test medication for their removal on an adequate basis, it is the opinion of the writer that the results of treating even two animals are worth placing on record.

Effects of Rumen Flukes on Livestock

In sheep fatalities resulting from acute infection with *Cotylophoron cotylophoron* are said to be very high. In South Africa, LeRoux[2] reported 30 and 50% death losses from flocks of 270 and 300 animals, respectively. In India, Bawa[1] recorded losses of 80 to 90% of the infected flocks. In India, Mudalier[6] observed fatal enteritis in infected goats.

Losses in cattle may result from infection with rumen flukes. In India, Maqsood[4] reported a case of acute paramphistomiasis in a cow 2½ years of age. This animal died as a result of the infection 14 days after admission to the clinic. Pande[10] recorded an outbreak of paramphistomiasis, later thought to be cotylophoronіasis[6], in cattle in which 30 to 40% of the cases terminated fatally within 15 to 20 days after the onset of a severe and fetid diarrhea accompanied by rapid emaciation. In France, Guilhon and Priouzeau[3] observed clinical paramphistomiasis in cattle. They reported the disease was most severe in young animals and that it often resulted in death. Mönnig[8] stated that purging occurred in both cattle and sheep as a result of amphistomiasis.

The clinical symptoms reported for acute amphistomiasis, caused by young flukes migrating in the intestinal mucosa of the small intestine, are depression, loss of appetite, general weakness, submaxillary edema, a fetid and persistent diarrhea beginning about the third day after the onset of symptoms, emaciation, and a high death rate.[1, 3, 4, 6]

As reported by various investigators, the general pathological picture found on postmortem examination was rather constant in all the animals dying from acute amphistomiasis. Large numbers of immature flukes embedded in the mucosa of the intestine and abomasum caused severe enteritis, congestion, many petechiae, and an acute catarrhal condition. In chronic cases there was marked infiltration of fluid into the pericardial sac, thorax, and celom, together with edema of the mediastinal, mesenteric, subcutaneous, and submaxillary tissues[3]. Numerous adult flukes in the rumen resulted in edema of the folds, destruction of the corneal layer, and chronic gastritis; emaciation occurred in spite of adequate nutrition.[3]

Preparation of Hexachloroethane-Bentonite Suspension

The hexachloroethane suspension was prepared as follows: 500 grams of commercial hexachloroethane ground so as to pass through a 60-mesh-to-the-inch screen, 50 grams of bentonite, and one-fourth to one-half teaspoonful of white flour were added to water (about 750cc) to make 1,000cc of suspension.[7, 8]

The drug was given by means of a metal dose syringe.

Trials with Hexachloroethane Against Paramphistomum

The first observation on the effect of hexachloroethane on rumen flukes was in a family milk cow which was kept in a small poorly drained pasture. In the early winter, the owner reported that the animal had declined in weight and milk production, notwithstanding an adequate ration of commercial feed. A fecal examination showed 252 eggs of the rumen fluke per gram of feces, indicating a heavy infection of adult flukes. Specimens of these flukes were not available for identification, but it is likely they were a species of *Paramphistomum*, judging by the morphology

*Parasitologist, Zoological Division, Bureau of Animal Industry, Agricultural Research Administration, USDA.

of the eggs. The cow was given three doses of 120cc each of hexachloroethane-bentonite suspension on consecutive days, making a total of 180 grams of the drug. Examination three days after the last treatment revealed only two eggs per gram of feces. Subsequent examinations, up to one year, failed to show recurrence of the rumen fluke even though the cow remained on the same pasture. Inasmuch as there was no marked physical response by the cow to the treatment and, presumably, subsequent expulsion of the flukes, it is not likely that the loss of weight and decline of milk production were due to the adult rumen flukes which she harbored at the time of medication.

The second observation was on an old ewe which suddenly developed symptoms of parasitism and was given 30cc of hexachloroethane suspension. She died within 24 hours of treatment; the time when death occurred during the night was not ascertained.

Of the 168 adult rumen flukes *(Paramphistomum microbothroides* Price and McIntosh, 1944, J. Parasitol. 30:9) collected from the region of the reticulorumenal orifice, 140 were dead and showed signs of degeneration, as indicated by their grey color, whereas the remaining 28 were alive and showed the pink color characteristic of this species. Young rumen flukes or morbid changes that are said to be associated with them were not seen. There were present, however, in addition to the adult rumen flukes, approximately 2,700 stomach worms, *Haemonchus contortus,* most of which were alive, and 296 liver flukes, *Fasciola hepatica,* of which 28 were dead in the liver and 68 in the small intestine. The stomach worms and liver flukes, rather than the rumen flukes, were considered as being responsible for the death of this animal.

Discussion

Since acute amphistomiasis is said to be a highly fatal disease in ruminants, it is important to prevent outbreaks of this parasitism that might result in severe losses. In regions where this fluke is known to occur, and where it cannot be controlled by eradicating the snail intermediate host, epizootics could be prevented by medicating affected livestock at such intervals as necessary, according to the ecology of the parasite and its intermediate host, to kill the adult flukes thereby preventing massive infections of young flukes which produce the acute form of the disease.

Carbon tetrachloride has been used with some success in the past for killing the adult rumen flukes in sheep. The doses required are large, however, ranging from 7cc of carbon tetrachloride in 13cc of sweet oil[1] to 8 to 10cc in raw linseed oil.[3] These doses are high for sheep and may in themselves be rather toxic. The therapeutic dose of hexachloroethane which is within the limits of tolerance of cattle,[8] sheep,[7] and goats[9] gave promise of being a safe and useful amphistomicide.

Summary

1. Acute amphistomiasis caused by young flukes has been reported as a severe disease of ruminants; killing 30 to 40% of herds of infected cattle and up to 90% of flocks of infected sheep. Goats also succumb to the effects of these flukes.

2. The morbid picture is rather constant, with large numbers of young flukes in the abomasal and duodenal mucosa, severe inflammation of the affected regions, accumulation of fluid in the body cavities, edema, anemia, degeneration of adipose tissue, and hypocalcemia.

3. Hexachloroethane - bentonite suspension removed all the rumen flukes from one cow, the results being based on pre- and post-treatment egg counts, and most of them from one sheep, when given as a drench at the rates of 120cc on each of three consecutive days and 30cc in a single dose, respectively.

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The Public Health Veterinarian*

By J. E. SCATTERDAY, D.V.M.**
Jacksonville, Florida

THE Florida State Board of Health established a Veterinary Public Health Program, January 1948, in the Bureau of Preventable Diseases, directed by R. F. Sondag, M.D. Veterinarians were classified as such for the first time in the state merit system. This classification is patterned after that of the U. S. Bureau of Animal Industry and definitely fixes the qualifications and compensation for each grade.

The public health veterinarian concerns himself with those diseases of animals which are communicable to man—their epidemiology and their control. This control is exercised generally in two major divisions—the control of the disease in the animal and the inspection of food products of animal origin, in the main, milk and meat.

To discuss these two major phases of veterinary public health in Florida it may be advantageous to discuss the different diseases which have been encountered in the past year.

Rabies Control Ineffective

Rabies was of first importance with 394 positive of 1134 dogs' heads examined in 1947, and 1434 antirabies treatments having been given to persons. In 1948, January through September, there were 225 positive for rabies of 835 specimens examined, and 1137 persons were given antirabies treatment. These figures for human prophylactic treatment comprise only those antirabic treatments furnished and administered by the Florida State Board of Health and its associate county health departments. They do not include antirabic treatments given by private physicians.

Rabies was of first importance

Since Florida has no uniform rabies program, but only the regulations adopted by the various municipalities with varying degrees of enforcement, we of the board of health felt that by better diagnostic methods and by improving our laboratory facilities—(1) better staining, (2) differential diagnosis from other virus diseases and (3) mouse inoculation from neurorrhyctes hydrophobiae negative specimens—we could be of more service in combating rabies. This, of course, does not eliminate the clinical diagnosis of the practicing veterinarian nor take the place of a complete history of the dog or other animal which did the biting as an aid to the physician in determining whether a person should receive the prophylatic treatment.

An informative program has been carried out in most of these counties where rabies has been reported. This program consists of pamphlets for distribution through the local county health departments; films furnished by the U. S. Public Health Service shown to schools, civic organizations, and groups such as the Parent-Teachers Association and others. Rabies has been discussed with the county commissioners, civic clubs, and local health departments. When rabies occurred in wild life the State Board of Health has coöperated with the United States Fish and Wildlife Service and the State Fresh Water Fish and Game Department in a trapping program. In Leon County more than 600 foxes were accounted for in approximately 90 days. This seems to have been effective in stopping fox rabies in that county.

The number of dogs vaccinated during the year has as yet not been tabulated, but in our animal report we hope to be able to give a fairly accurate report on this.

Brucellosis Control Program

Brucellosis of which 57 human cases have been diagnosed and reported for 1948 presents a major problem. These 57 laboratory confirmed cases of brucellosis, of course, do not present a true picture of the prevalence of the disease in Florida. We feel that many more cases of brucellosis occurred but were not confirmed by the State Board of Health laboratory and are not reported.

Our method of attack has been to encourage the adoption of local milk ordinances by cities in Florida, requiring that herds produc-

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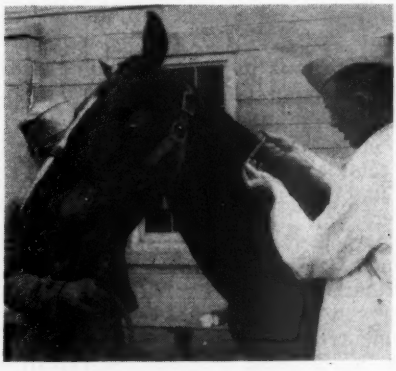
**Public Health Veterinarian, Florida State Board of Health.

ing milk for sale in those cities adopt some type of brucellosis program recognized by the U. S. Bureau of Animal Industry and the Florida Live Stock Sanitary Board, which will eventually lead to a herd free from brucellosis. This plan can be adopted only by those dairies which produce milk for pasteurization. The Florida milk laws specifically require that milk to be consumed raw must come from cattle free from brucellosis. Thought must also be given to the fact that brucellosis is spread from animals to man through direct contact and that the disease is an occupational one with dairymen, farmers, abattoir workers and, of course, veterinarians. Therefore a program to identify those animals which react to the brucellosis test and to protect the negative animals is essential.

Lay county sanitarians have been instructed, while making routine sanitary inspections in rural areas, to inquire into the status of the family milk cow, to encourage the owner to have her tested for brucellosis by his local, federal, or state veterinarian. They have also been instructed to assist the veterinarian in this examination to the extent of arranging this work and actually assisting when possible. These sanitarians are given credit for this work on their daily report to the local health officer.

Equine Encephalomyelitis

Outbreaks of equine encephalomyelitis are investigated. Specimens are collected and forwarded to the virus laboratory at Montgomery,



Information and suggestions have been disseminated to encourage the vaccination of susceptible animals against the disease

Alabama, and to the National Institute of Health, Division of Pathology, for diagnosis. In case the disease is diagnosed, the local county sanitarians, in cooperation with the Florida State Board of Health, Bureau of Entomology, spray all buildings, corrals, etc., on the premises with D.D.T. to control the insect vectors. Educational work through the local health departments and agriculture agencies has been done to encourage the vaccination of susceptible animals against this disease.

Veterinary P. H. Laboratory

A veterinary public health laboratory in the Florida State Board of Health laboratory is now getting into operation. This branch of the laboratory will assist in diagnosis of diseases of animals communicable to man. The success of a laboratory of this kind depends upon the veterinarians of Florida. Unless they use it and submit a sufficient number of specimens it will not warrant the expenditure for qualified personnel and equipment. It is hoped that a laboratory of this kind may materially aid the practicing veterinarian as well as those engaged in public health work.

The laboratory section of the State Board of Health has developed a rapid test for tularemia similar to the test for brucellosis. Educational efforts are being made to control this disease. To date in 1948 only 14 cases have been reported and confirmed by laboratory diagnosis.

Ornithosis and Psittacosis

Birds of the psittacine family are prohibited by federal law from being shipped interstate with but two exceptions, first, those destined to a zoological park or research institute; secondly, pets, not to exceed two birds which have been in the owner's possession for at least two years. For both of these exceptions Florida law requires a health certificate by a qualified veterinarian certifying the birds to be free from psittacosis and that the premises from which they come are free from the disease.

Milk Inspection

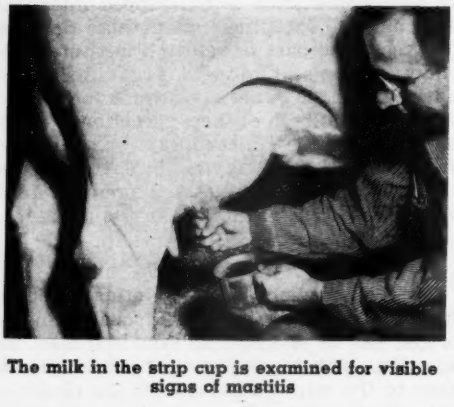
The second phase of veterinary public health, that of inspection of foods of animal origin, can be divided into milk and meat inspection. Milk inspection is of prime importance to the veterinarian in that the health of the cow is essential to good milk and without a healthy cow no dairy can be classified as Grade A. The Florida State Department of Agriculture, milk control section, and the Florida State Board of Health's sanitary code both require an annual tuberculin test of all cattle over six months of age before milk from the herd can be legally sold. As Florida does not raise its own dairy cow replacements and is an importing state, this is very important, since the replacements originate from all southern states and as far north as New York

and Wisconsin. The average life of the dairy cow in this state is 2½ years, therefore many cattle would not be tested if the testing were at three-year intervals as is the case of the

A tuberculin test is made annually

modified accredited plans. If Florida had a good meat inspection program these tuberculous animals would be found on post-mortem examination, but a large percentage of the unprofitable dairy cows are slaughtered under poor inspection or no inspection at all.

The brucellosis test is a requirement only for



The milk in the strip cup is examined for visible signs of mastitis

those dairies which sell their products to be consumed raw. That this disease may be contracted by direct contact with the infected animal is not taken into consideration and we feel that a good percentage of all brucellosis reported here in Florida may be due to this mode of inoculation rather than from ingestion. Therefore, we of the State Board of Health recommend that all herds whether the milk is to be consumed raw or pasteurized should operate under some approved type of brucellosis control which, in the end, will give the dairymen a negative herd.

The county sanitarians, who are responsible for the county and municipal milk inspection, keep a record of all tuberculosis and brucellosis tests and should notify the dairymen in advance when such tests are due—and enforce the regulations by degrading or revoking the permits to sell milk of all dairies that become delinquent. These men also watch for abnormal milk. This is purely a lay job and consists of observing the prestripping in the strip cup and the filter disks in the strainer for visible signs of mastitis. Cows giving abnormal milk are to be excluded from the milking line and remain out until the milk is normal.

Meat Inspection, Good, Poor and Nonexistent

Meat inspection in Florida may be divided into three classes, (1) federal or B.A.I., of which we have four establishments, (2) state inspected abattoirs of which 16 establishments have met the requirement of the State Live Stock Sanitary Board for construction, sanitation, ante-mortem and post-mortem inspections and (3) municipal or county inspection. In slaughtering establishments below federal or state inspection level, these vary from fair in inspection to none, and from abattoirs fairly well constructed and operated, to an oak tree in the woods. This condition depends upon the local ordinance and the degree of enforcement. It is the intention of the board of health to eventually bring this type of slaughter up to the level of state inspection as conducted by the Florida Live Stock Sanitary Board.

Veterinarians in Public Health Service

The Florida State Board of Health, exclusive of the central office in Jacksonville, now consists of 62 county health units, leaving only five counties in the state without such units. The units are manned by 43 full time health officers and their numerous assistants, 131 sanitary officers, 21 sanitarians, five sanitary engineers on a county level and six full time veterinarians. As the value of those veterinarians in the various phases of public health become recognized, it is hoped that all the larger health units, and combinations of some of the smaller counties will see fit to add this type of public health service. It is the present responsibility of those full time public health veterinarians and practitioners who engage in part-time meat and milk inspection to demonstrate to the public the necessity for veterinary service to provide wholesome, nutritious foods of animal origin and to protect from diseases communicable from animals to man.

Technic of Direct Blood Transfusions in Large Animals*

By W. M. COFFEE, DV.M.
La Center, Kentucky

THE USE of whole blood in large animal practice has not only proved to be of great value as a therapeutic agent, but adds to the prestige of the user. Whole blood is of value in cases of shock, severe hemorrhages, purpura hemorrhagic, phenothiazine poisoning, sweet clover poisoning, parasitism, anaplasmosis, icteric foals and various anemias.

We have not found it necessary to type blood in the ordinary farm horse or cow, even though a number of different types are found. In the thoroughbred animal over 50 different types have been demonstrated. Therefore, in the valuable thoroughbred it may be advisable to type. In the icteric foal the blood should always be typed, as the cause of this condition is an abnormal destruction of erythrocytes, believed to be due to a red blood cell factor comparable to the Rh factor in human blood. In nearly all tests conducted it was established in each case that the serum of the dam agglutinated the erythrocytes of the sire, and that the serum of the sire did not agglutinate the cells of the dam. In some instances sufficient antibodies have been acquired through the mare's colostrum to start abnormal red cell destruction in the foal. Therefore, in no case of icterus in the foal should the dam be considered as a blood donor.

It is not necessary to type blood in the ordinary farm horse or cow. In a valuable thoroughbred it may be desirable to type the blood

My methods and technic of a blood transfusion in horses and cows, which can be used either at the hospital or in the barnyard, is as follows: Place a piece of glass rubber surface tubing 3½ feet long on each end of a Shikles' hog cholera syringe, first filling the inlet tube by placing the needle in a bottle of 10% sodium citrate solution. This is to exclude the air from the tube and to form an anticoagulant coating in the tube, to prevent coagulation of the blood as it enters the syringe. If the donor should be a work horse, put his harness on him; if a saddle horse, place the saddle on him. This seems to relax them and they stand much quieter during the transfusion. The same applies to a cow. Put her in her stanchion. Stand the two animals side by side working between them, using a 12-gauge, 2½ inch needle in both the donor and the patient, with the needle pointing up the jugular in the donor and down the jugular in the patient. Have an assistant put pressure below the needle on the jugular of the donor, and hold the needle and tubing in line with the jugular. The citrate may be pumped through the syringe and tubing and wasted before connecting the tubing to the needle in the patient. The operator using the syringe can determine the amount of blood transfused by counting the number of syringe fulls.

Have at hand a bucket of cold sodium hypochlorite solution to wash the tubing and syringe, immediately following disconnection from the donor. This will prevent coagulation in the tubing and syringe. The secret in the direct blood transfusion is to fill the tubing with citrate and exclude air from the blood. This prevents coagulation in tubes and syringe while giving transfusions.

In the event the donor and the patient cannot be placed side by side, the blood may be collected in a bottle containing the necessary amount of citrate, and given by gravity. In case of icteric foals, where large quantities of transfused blood are required, it may be necessary to place a needle in the opposite jugular of the foal, thus allowing him to bleed at the same time he is receiving blood.

*Presented at the 19th annual meeting of the Florida State Veterinary Medical Association, Oct. 24-26, 1948, Orlando.

Problems of

Swine Production

Sound Advice on These Problems is Profitable to the Swine Raiser and to the Veterinarian

Presented at the 19th annual meeting of the Florida State Veterinary Medical Association, Oct. 24-26, 1948, Orlando.

By W. M. COFFEE, D.V.M.
La Center, Kentucky

ON THE subject assigned, "Swine Problems and Production," I shall not attempt to go into the details of any particular phase, but will touch on several problems confronting swine production, and the veterinarian's interest therein. The price of swine for the past few years has brought an increased demand for professional veterinary services and for advice, and in many instances as a result of courteous sound advice, the demand for veterinary services has been on the increase. When veterinarians are called upon to assist feeders and breeders in their feeding and breeding problems, they not only build good will and future business, but improve their practices, since a successful client is the more profitable one in every respect.

Statistics show that 35% of all pigs die before weaning. Sixty per cent of this loss can be prevented by proper herd management and feeding during the gestation and lactation periods. Any information veterinarians give their clients that reduces this loss results in greater profit for both parties. We know that a pig weighing three pounds at birth seldom gets killed or dies from any cause other than disease. Therefore, proper nutrition during gestation is very essential to a reduction in the loss of young pigs.

A protein supplement constituting approximately 16% of the ration and including a liberal amount of animal protein (preferably tankage) supplying the essential amino acids, together with ground alfalfa and yellow corn has proved a satisfactory brood sow diet. But one must not overlook adding the essential mineral elements and vitamins to this ration; especially calcium, phosphorous, salt, an iodide and vitamins A, B and D, through cod liver oil and yeast.

This feeding practice will solve the problems of jittery and undersized pigs at birth. The so-called jitter bug or dancing pig makes a rapid recovery when injected with 5 to 7cc of calcium, phosphorus and glucose solution and given plenty of milk and sunlight. But it is a lot better to prevent these nervous attacks by proper nutrition of the sow during gestation.

Agalactia.—The proper diet of a sow during gestation will, to a large extent, prevent a failure of the sow to furnish an adequate amount of milk. After agalactia sets in, the pigs soon starve. Soaked oats is a milk-producing diet, but is too slow to save the pigs of a sow already affected. As a medicinal agent to stimulate the flow of milk I use 5cc of posterior pituitary extract and 5cc anterior pituitary extract simultaneously. If there are any signs of constipation in the sow, I give her four to eight ounces of mineral oil through the stomach tube. Always see that the diet is properly balanced and the sow has access to plenty of clean water.

Anemia.—Sucking pigs, particularly winter pigs, are apt to suffer from iron deficiency. Regardless of what sows are fed, iron is not transmitted through their milk. Iron, no doubt, is one of the essentials for preventing pig anemia, but there are other elements that play an important part in it. I am of the opinion that many pigs suffer from anemia shortly following birth, to an extent that although they do not die, they are stunted before they are able to acquire these elements unassisted. Iron and copper may be supplied to these little fellows by painting the dam's teats, after farrowing with an iron solution, or by giving each pig an antianemia tablet. The placing of sod in farrowing pens for the pigs to lie on or root, is also of value, but somewhat less dependable than direct dosing. In every instance, I recommend putting the pig out on clean soil with plenty of sunlight, soon after farrowing, if the weather will permit it. All of these recommendations assist in starting a baby pig on the way to market.

If anemia has already developed in the pig when I am called, I inject intraperitoneally 1cc to 3cc of CA-CO copper and dispense anemia tablets to be given at three to five-day intervals. Do not be surprised if a few sick pigs die, but remember the advantages of prevention, and begin treating the entire litter at the first appearance of the disease in any member of the litter. In my opinion the subclinical or unidentified form of anemia is responsible for many other diseases, as it results in lowered resistance of the pig to any and all diseases.

Selection of Brood Sows.—Much can be said about the selection of brood sows for a good breeding herd. Other than being from large litters, of good size and conformation with a uniform udder, the history of the herd and the farm from which the brood sows come is of importance. Many fine looking sows are carriers of disease. I have noticed that the pigs of certain sows always are the first in the herd to come down with enteritis, pulmonary disturbances, erysipelas, etc. Such infections are probably carried in chronic lesions in the sow, or perhaps in the gall bladder as demonstrated by some research workers. On farms where these conditions are present a farmer should be advised not to keep for breeding, any of the gilts from litters that break early. In fact, no gilt that has suffered an illness should be retained for breeding.

Never overlook brucellosis in a herd. It is an important cause of baby pig mortality. The disease has many perplexities. Some sows will abort once and carry subsequent litters, to term. Some will react to the agglutination test and never abort. The agglutination test is a good herd guide to be governed by, and purchased boars should have at least two negative tests, 30 days apart, before being added to a clean herd of brood sows. Vaccination of gilts for brucellosis is not generally practiced. There is experimental evidence that it is of little value. Nevertheless, on infected farms good results have been obtained and pigs successfully raised where gilts were given 5cc of Strain 19 vaccine, at six or seven months of age. Unvaccinated gilts retained in the infected herd as controls later became infected with pig losses as a result.

Parasite Control.—All farms where hogs have been previously raised or fed are infected with parasites and unless the McClean County system is followed closely, most pigs begin to suffer from both internal and external parasites at about weaning age. Of course it is better to prevent the condition by sanitation, but if it occurs, I find oil of chenopodium, given in individual doses, to be the most effective vermifuge for pigs affected with ascarids, which is the common intestinal parasite at this age. If the pigs are drinking slop, phenothiazine solution, 28 grains to 100 pounds body weight, is my second choice, and for hogs of uneven weights on dry feed, a 1% sodium fluoride diet for 24 hours is very effective. I have found nothing better for controlling external parasites than 1½% DDT in a 5% lime-sulphur suspension in water. Spray hogs and houses with this mixture once per month. This mixture not only destroys the lice, but prevents swine pox which is transmitted by the louse. It is also effective for mange and other skin ailments.

Observations on Canine Leucemia

By RAY H. BRADBURY, D.V.M.
Mount Vernon, Washington

A SEVEN-YEAR-OLD German Shepherd dog was presented for examination to determine the cause of loss of weight and attacks of severe dyspnea. These attacks consistently followed slight exercise. His general condition was considered fair on casual observation. However, on palpation of his body, a generalized enlargement of lymph glands was obvious beneath his long hair. The popliteal lymph glands were readily palpable; being about the size of two walnuts.

The dog was hospitalized for two weeks and many tests and observations were made. The spleen was palpable and of solid consistency. Concurrent with the glandular enlargement were symptoms of a mild gastroenteritis. The daily temperature varied from 101 to 102.4°F. The odor of the feces and the mouth were not unlike that found in intestinal distemper. The feces were soft and sometimes mucoid, but never bloody. The mucous membrane of the mouth was pale and consistently spotted with transudate blood. The molars were partially covered with a dark film. The sclerae were congested; having a rust-like appearance. On the 12th day the dog was extremely depressed; the dyspnea was marked, continuous and

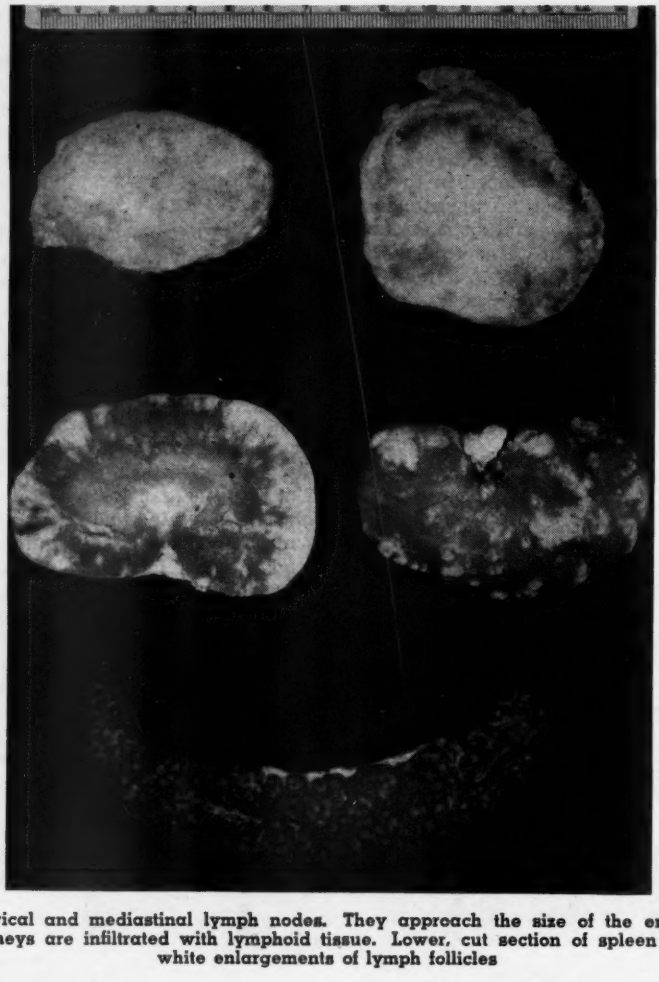


Fig. 1—Top, cervical and mediastinal lymph nodes. They approach the size of the enlarged kidneys, middle. The kidneys are infiltrated with lymphoid tissue. Lower, cut section of spleen revealing grey-white enlargements of lymph follicles

distressing. He had to be dragged from the kennel for treatment. On the 15th day, signs of approaching death were obvious and he was given a lethal dose of nembutal and taken to the morgue at the Western State Hospital for autopsy. Dr. Wm. Dublin, pathologist, and formerly associated with the Mayo

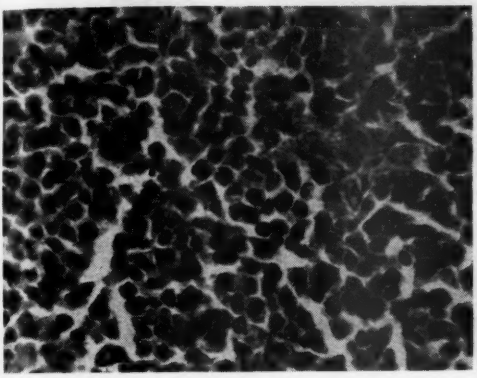


Fig. 2—Photomicrograph of lymph node X400. The cell pattern is similar to that found in leukemia of man

Foundation, had expressed his desire to compare the pathological changes in leucemia of dogs with that of man. His convincing autopsy report is appended.

Dr. Dublin is an eminent neuro-pathologist and was particularly interested in the changes in the nervous system. I am profoundly obligated to him for this unusually complete and extremely informative necropsy report.

Gross Examination

Lymph nodes: Enlarged, reaching a diameter of 6cm, in practically all regions of the body, including submaxillary, cervical, mediastinal, axillary, inguinal, and abdominal. Discrete, moderately firm and semi-rubbery, with cut surface resembling fish flesh, being grey-white, slightly gelatinous in appearance. Areas of hemorrhage and many areas of degeneration seen. Capsules of nodes intact.

Heart: Not unusual.

Lungs: Scattered areas of grey hepatization of bronchopneumonia.

Spleen: 340gm Firm, red, with grey-white enlargement of lymph follicles, which average 2 to 3mm, and are thickly distributed.

Liver: 1460gm Yellow-brown, with very fine whitish flecks of infiltration.

Esophagus: Not unusual.

Stomach: Folds prominent and thickened.

Intestine: *Dipylidium caninum* present. Folds thickened, and in the submucosa miliary whitish nodules which are suggestive of hypertrophy of lymph follicles. Hemorrhagic enteritis not outstanding, although a few superficial erosions are seen.

Adrenals: Not unusual.

Kidneys: Each enlarged; on cortical areas multiple, firm, white, raised plateau-like areas of infiltration. On cut section, these are seen to extend inward through a portion of the parenchyma, and there are further, segregated (buried) areas of whitish infiltration deep within the kidney substance.

Bladder: Not unusual.

Prostate and Testes: Not unusual grossly, although the prostate seems a little white and firm.

Thyroid and Aorta: Not unusual.

Spine: Not sectioned.

Brain, Cord and Brachial Nerves: Not remarkable grossly.

Microscopic Examination

Blood: Hgb. 50% RBC 3.2 million. WBC 20,100. Lymphoblasts 15, Pathologic lymphocytes 5, Polymorphs 55, Lymphocytes 126, Monocytes 6. Nucleated reds 2; total cells counted 209. Sedimentation rate 49mm. in one hour. Kolmer. There are numerous lymphoblasts, with large nuclei, plus scant cytoplasm, and especially, prominent, multiple nucleoli.

Heart: Small scattered areas of infiltration with lymphoblasts.

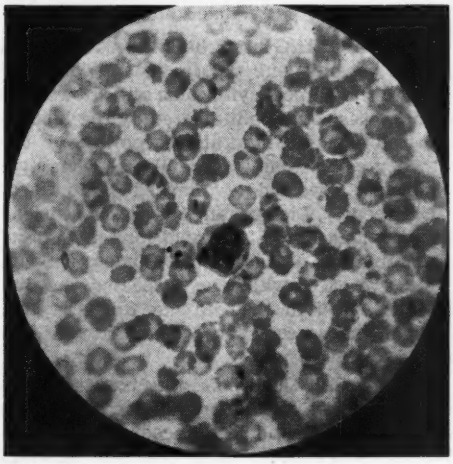


Fig. 3—Photomicrograph of stained blood smear. Note typical lymphoblast in center of field

Lungs: Hypostatic pneumonia.

Spleen: Lymphoblastic hyperplasia in follicles.

Liver: Periportal infiltration of leukemia.

Lymph nodes: Lymphoblastic proliferation.

Adrenal: Normal.

Kidney: Leucemic infiltration.

Stomach and Intestine: Leucemic hyperplasia of mucosa and of follicles of submucosa; muscle spared.

Pancreas: Normal.

Prostate: Leucemic infiltration nearly replaces stroma between glandular acini.

Brain and Cord: Spared; there is one cluster of lymphocytes in meninges of cord which is not definitely leucemic.

Lead Poisoning in Dogs

By ELIHU BOND*, D.V.M.
New London, Connecticut

and ROSA KUBIN, Ph.D.,
Waltham, Massachusetts

LEAD POISONING must be known to exist in dogs, yet only one report, that of Mitchell[1] describes the clinical symptoms and shows that the diagnosis could be indicated by the laboratory findings: anemia and stippling of the erythrocytes. Post-mortem analysis of the liver in his case revealed a "marked" amount of lead. Recently, Lieberman[2] has described lead poisoning as a cause of convulsions and found it in a number of cases post mortem; he describes two cases where he suspected the disease ante mortem. He did not find stippling in all of his cases where the blood was examined.

Experimentally lead poisoning has been produced in dogs[3-8]. The symptoms seen were paralysis of the hind legs, incoördination, excitability, convulsions and tremors. Anemia and stippling were seen[4,5,6] but not always.[7,8]

We are reporting nine cases. Our first two cases (No. 1 and 2) were discovered accidentally and led us to look for the condition in the others.

Case 1.—A spayed Cocker Spaniel 10 months of age, was examined for a non-specific abdominal ailment. She showed anorexia, constipation, and listlessness. She was given an enema and appeared improved. Two days later she was again admitted to the hospital. She was weak, dehydrated, anoretic, and her temperature was 100.8° F. Leptospirosis was suspected and she was given saline-dextrose and penicillin. The urine showed slight albuminuria, an increased urobilinogen and a trace of bile. On the second and third days, the temperature dropped to 98.8° F. but the treatment was continued. A blood and urine examination at this time revealed a hyperchromic anemia, leucocytosis, basophilic stippling of erythrocytes and a thrombocytosis (see table). The urine showed some albuminuria.

This dog died four days after admission. Autopsy revealed a mild enteritis. Analysis of a portion of the liver sent to an analytical laboratory,[12] showed 15 parts per million (ppm) of lead. Urine mailed previously contained 0.8 ppm.

Case 2.—Cocker Spaniel, 15 months of age, was examined showing anorexia and moaning from abdominal pain. She was given milk of magnesia and an injection of atropine. She improved and ate the next day. Four days later she returned with the same colicky symptoms, and a temperature of 102.6° F. She was given milk of magnesia and atropine as before and also penicillin, but showed no improvement and nembutal was used as a soporific. Urinalysis was not remarkable except for a trace of sugar and an orange precipitate, on addition of Erlich's aldehyde reagent. The pain was gone the next day but the dog appeared depressed. She was observed for two more days, then a blood examination was made. It revealed a leucocytosis, hyperchromic anemia, and a marked number of normoblasts which all showed basophilic stippling (see table). A diagnosis of lead poisoning was made and a high calcium

*Now Administrator of Animal Hospitals, Chicago Professional Colleges, University of Illinois.

diet was started. She improved markedly and gained weight. She had colic again seven months later and the high calcium diet was instituted but unfortunately was not kept up and six days later she had another relapse. Calcium was given intravenously and she vomited up a mass of hard fat and swill. She ate the following day but had a colicky attack five days later. The high calcium diet was started again. Since then the owner has changed the diet to another dog food and the dog has been symptomless for eight months.

Case 3.—A spayed mongrel Shepherd, 10 months of age, had been examined on various occasions for abdominal pains, vomiting, anorexia, and restlessness. At the third visit a stained blood smear showed many normoblasts. A complete blood examination was therefore indicated and showed leucocytosis, hyperchromic anemia, marked number of normoblasts—all of which showed basophilic stippling—a reticulocytosis and thrombocytosis (see table). Her colicky attacks have subsided after treatment with a high calcium diet.

Case 4.—A female Cocker Spaniel, 3½ months of age, had been treated for "diarrhea and fits." An examination of a stained blood smear showed a marked number of normoblasts. Complete blood examination was consistent with the diagnosis of lead poisoning. The leucocyte count was 54,100, the anemia was markedly hypochromic, all normoblasts and more than 10% of the erythrocytes showed basophilic stippling. A few Howell-Jolly bodies were seen. Platelets were very numerous (see table). The dog was put on a high calcium diet along with iron and vitamin B complex therapy. There has been a cessation of the fits and the general condition of the dog has improved considerably since treatment began.

Case 5.—A female mongrel Fox Terrier, seven months of age, had been vomiting and passing formed dark boluses of feces for four days. There was severe abdominal pain but the abdominal walls were not tense. The temperature was 99.6° F. Fecal examination revealed whipworm eggs. Atropine, gastric sedatives and sulfathalidine were given. There was no

TABLE I.—SHOWING THE RESULT OF BLOOD EXAMINATION IN MISCELLANEOUS CASES OF SPONTANEOUS LEAD POISONING IN DOGS.

Case	RBC	Hg.	Ht.	CI	MCV	WBC	Neut.	Unseg.	Lymph.	Mono.	Eos.	Norm.	Plate.	Stippling	Remarks
1	4.78	15	—	1.4	—	16,500	77	4	15	0	0	1	Incr.	+	
2	3.97	10.4	42	1.17	120	13,150	84	5	9	0	0	68	Norm.	++	
3	5.24	11.2	45	1.03	85	25,150	80	3	16	0	1	79	Incr.	+++	Polychromatophilia
4	3.31	6.5	22	0.88	67	54,100	76	15	8	0	1	60	Incr.	+++	Polychromatophilia
5	3.70	7.3	29	0.89	79	25,700	62	9	21	3	0	163	Incr.	+++	Polychromatophilia
6	4.85	14	—	1.25	—	34,500	88	5	7	0	0	3	Incr.	+++	
6 wks. later	5.80	13.5	48	1.05	82	12.500	64	7	28	0	1	5	Norm.	++	
7	5.65	12.8	45	1.00	80	11,600	76	6	18	0	0	8	Norm.	++	Polychromatophilia
2 wks. later	4.49	10.5	36	1.00	80	7,300	81	5	14	0	0	12	Norm.	+++	Increased Polychromatophilia
1½ wks. later	4.23	12.0	35	1.25	87	11,400	67	4	24	0	5	1	Norm.	++	Increased Polychromatophilia
8	3.82	9.6	32	1.13	84	21,300	70	14	10	4	2	70	Incr.	+++	
9	3.12	7.0	24	1.00	79	18,700	68	21	11	0	0	80	Incr.	+++	Polychromatophilia

RBC—Red cell count in millions.
Hg.—Hemoglobin in grams.
Ht.—Hematocrit.
CI—Color Index.
MCV—Mean Corpuscular Volume in cubic microns.
Norm.—Normoblasts per 100 WBC.
Plate.—Platelets increased or normal number.

Stippling: + = Some stippling.
++ = Moderate stippling.
+++ = Marked stippling.

improvement the next day. Fluoroscopy revealed gas throughout the intestinal tract. The dog still vomited. Morphine, calcium gluconate, and fluids were given parenterally. A blood smear was made. A tentative diagnosis of lead poisoning was made because numerous normoblasts were present. The temperature dropped to 95° F. that night. Blood was taken, and euthanasia was performed. Blood examination revealed leucocytosis, hypochromic anemia, a very large number of normoblasts and marked stippling of the erythrocytes and normoblasts. There was also a thrombocytosis (see table). Autopsy showed a hemorrhagic duodenum and anterior jejunum. Analysis of the liver revealed 30 ppm. of lead, 40 ppm. of zinc and 40 ppm. of copper.

Case 6.—A four-year-old, male Cocker Spaniel showed abdominal pain for four days, was sensitive over the kidneys and vomited frequently. The temperature was normal. Leptospirosis was suspected. Albuminuria was demonstrated. The blood showed leucocytosis, hyperchromic anemia, a few normoblasts, basophilic stippling of the erythrocytes, and thrombocytosis (see table). Epsom salts was given. During the acute attacks that followed, calcium gluconate was given intravenously. Elimination of the lead from the bones was attempted by giving ammonium chloride. Vitamin B and liver injections were given. Blood examination, six weeks later, showed an increase in the number of erythrocytes, a decrease in the color index, decrease in the leucocytosis, and less stippling with an increase in the number of normoblasts (see table).

Case 7.—An eleven-year-old spayed mongrel had been dull for three weeks with a poor appetite, loss of weight, spells of diarrhea and no fever. Bismuth had no effect on the diarrhea. Palpation revealed no masses in the abdomen. Blood examination demonstrated a normochromic anemia, moderate leucocytosis, some normoblasts, all of which showed stippling with few of the erythrocytes showing stippling; polychromatophilia was seen (see table). Two weeks later the dog showed anorexia, convulsions, and emesis. The urine showed albuminuria, blood cells, renal epithelial cells, and casts. Blood examination revealed a severe anemia, increased number of normoblasts and marked stippling. Liquids were forced orally and parenterally and ammonium chloride was given. Ten days later, there was marked clinical improvement. A blood examination at that time showed an increase in hemoglobin, a shift in the anemia to a hyperchromic type with less normoblasts but more polychromatophilia (reticulocytes) (see table). Vitamin B, liver, and iron were given and three weeks later the dog was completely well.

Case 8.—A male Cocker Spaniel, one year of age, showed vomiting, listlessness, anorexia, and no thirst. This dog had been under the care of another veterinarian who had prescribed triple-arsenates, but it worsened and exhibited abdominal pains. By the time the dog was seen the abdominal pains were severe and the animal was markedly dehydrated. Fluids were given parenterally and intestinal protectives were supplied orally along with calcium. The dog appeared improved but had a convulsion the next day. He was given calcium parenterally but convulsions continued until he was given nembutal. The convulsions were marked by a doubling up and tensing of the abdominal and leg muscles but no frothing at the mouth was seen. Blood was examined and revealed a very marked hyperchromic anemia, leucocytosis, large number of normoblasts, with basophilic stippling seen in many mature erythrocytes and in all the normoblasts. Platelets were very numerous (see table). Calcium was given parenterally until convulsions ceased, then elimination of the lead was attempted with ammonium chloride. The dog had a few convulsions after that which were treated with calcium parenterally. Four weeks later he appeared to have recovered.

Case 9.—A male Dachshund, 10 months of age, was listless and showed inappetence for three weeks. He had had a number of convulsions and various veterinarians had examined him.

A diagnosis of encephalitis was made and the dog was given penicillin. He continued to vomit and have frequent convulsions. Liquids were given parenterally and finally nembutal was used to stop the convulsions.

A blood examination at this time revealed a very marked normochromic anemia, leucocytosis, many normoblasts and stippling in all the normoblasts and many mature red cells, with polychromatophilia and thrombocytosis (see table). Calcium gluconate was used alternately with ammonium chloride and after seven weeks the dog had recovered.

Analysis of Symptoms

The symptoms seen in lead poisoning are not unlike those seen in any gastroenteritis, including cases of leptospirosis and uremia which appear as a gastroenteritis. The onset is marked by inappetence which may be complete. Vomiting then occurs which is accompanied by either diarrhea or constipation, or may exist alone. Colic is an important symptom and is shown by moaning and grunting

or, in a mild form, by restlessness; the dog can't seem to find a comfortable position. Convulsions occurred in four of the nine cases. They ran the gamut from barking fits to those resembling encephalitis with the frothing at the month and champing of the jaws. Apparently they are analagous to the lead encephalopathy seen in human cases[3]. They also indicate how diverse are the causes of "fits" in dogs.

A high calcium diet is fed and where indicated calcium is given intravenously

No "lead line" of the gums was seen in any of our cases.

Analysis of Laboratory Findings

Urine was examined in only four cases. However, albuminuria was present in three of these. In only one case was increased urobilinogen excretion observed (case 1). These findings are not conclusive; however, in human cases, albuminuria and increased urobilinogen are seen in an extremely high number of cases.[9]

The significant laboratory findings are the blood changes. The anemia and stippling were seen in all of our cases. The anemias were of different types. Four of the nine were hyperchromic, three were normocytic and two were hypochromic. All of the normoblasts seen contained basophilic stippling but the stippling was not consistent in the mature erythrocytes. Normoblasts were present in all the blood specimens. The range was from one to 163 in 100 white blood cells. They were so numerous at times that a casual glance at the smear under high power indicated a marked anemia and allowed a tentative diagnosis of lead poisoning to be made before the complete blood report was received. The leucocyte count was never below 11,600 and even ran as high as 54,000. There was a shift to the left of the nonsegmented neutrophils. Seven cases showed an increase in the number of platelets; in the other two cases, they were normal in number. Polychromatophilia was marked in five cases. Howell-Jolly bodies were seen in three cases.

Although we realize that only chemical demonstration of a toxic amount of lead in the tissues or excreta is conclusive evidence of lead poisoning, we feel that diagnosis of lead poisoning may be justified, if the clinical symptoms, described in the foregoing are accompanied by the characteristic blood picture.

Treatment

The metabolism of lead is related to that of calcium. Anything that helps deposit calcium in the bones will do the same for lead.[9] With this in mind, lead is pushed into the bones where it is harmless, if it remains there.

A purge is always indicated in a poisoning and magnesium sulfate is preferred. It is not only effective as a purge, but also may precipitate the lead as the insoluble—and therefore, harmless—lead sulfate. Calcium is then given parenterally. In lead colic it is especially efficacious if given intravenously because it pushes the lead toward the bones and also relieves the spasm due to the action of the calcium on the smooth muscles.[9] A high calcium diet is then initiated. One to two grams of dicalcium phosphate preferably with vitamin D are given daily along with plenty of milk, etc., until the dog is symptom free. At that time deleading of the bones may be started. It must be borne in mind that any illness, or change in metabolism of the animal may bring about spontaneous deleading with a subsequent increase in the blood lead and symptoms of lead poisoning. Any infection, disease, acidosis, large doses of alkali, lactation, starvation, etc., may bring this about.[9] Therefore the dog must be kept in perfect health while deleading is going on, or the deleading must be postponed until the dog is well again. Ammonium chloride or nitrate is used as a deleading agent. Giving either of these drugs increases the excretion of lead in the urine. Potassium iodide is also used but its action is not as dependable nor as prolonged, as that of the ammonium salts.[9] Two or three grams of ammonium chloride may be given daily to a dog of average size. This must be accompanied by a low calcium

diet. Deleading should continue for a month and should be suspended whenever toxic symptoms appear. At such times, the high calcium diet should be started and kept up until the animal is symptomless. During recovery, iron, liver and vitamins should be given to hasten it.

Source of Metal and Toxicology

In six of the nine cases the owners were painting the outside of their houses. Indoor paints do not contain lead.[13] Outdoor paints, especially if they are a shade of white, usually contain white lead. Not only is white lead used for its color, but it is also mixed into other colors to give the paint more durability. A seventh case may have been caused by the dog eating putty that contained white lead. Most putty does not contain lead since it is too expensive, but the dog ate some putty and there is a possibility that it contained lead—no other source of the metal could be incriminated. In the other two cases and in others that we have seen at autopsy, no clue as to the source of the metal could be found. We have seen high concentrations of lead in the liver and yet the owners could not think of anything that could have caused the poisoning. In our autopsy cases, no arsenic was found, so that we were able to rule out lead arsenate as a source of the metal. In a number of cases, the lead has been accompanied by high values for copper and zinc. It is possible that corrosion of the brass piping in the houses may account for the other metals.

Some authorities, not all, are of the opinion that lead is a cumulative poison.[12] Inhalation of lead particles is the most toxic means of becoming poisoned, while the ingestion of the metal is next toxic and the most common.[9] We have not had any lungs analyzed for the metal; and we are not in a position to state that any of our cases received the metal by inhalation. About the only common thing that might be inhaled is leaded gas fumes or exhaust from engines using anti-knock gas. On the other hand, large quantities of lead may be ingested, but large quantities are also excreted in the feces and a large amount that is absorbed is excreted in the bile.[9]

We do not know the upper normal limits of lead in the internal organs of animals. Even in humans, values range widely. However, the upper normal human limits accepted by some authorities are as follows: blood, 0.8 parts per million (0.08mg per 100cc), urine, 0.08 ppm. (0.08mg per 1000cc[9]), liver, 5 ppm.[12] However, an amount above those limits does not mean that poisoning exists; the toxic limits vary because of individual susceptibility. Cases 1 and 5 showed marked amounts of lead in the liver and also showed the clinical symptoms of poisoning. Case 1 had 15 ppm. in the liver and also showed 0.8 ppm. in the urine.[15] Both findings are markedly above the human toxic limits. Case 5 showed 30 ppm. of lead in the liver[12] and clinically seemed worse off than the others.

Experimentally, dogs fed enough lead to give them 2.0 ppm. in the blood and 8 ppm. in the liver did not show any symptoms, whereas dogs fed enough lead to give "mild" symptoms showed 6.0 ppm. in the blood and 32 ppm. in the liver.[8] Perhaps the acuteness or chronicity of the poisoning and the types of lead salts ingested might explain the marked difference between the experimental and spontaneous cases.

Comments

Lead poisoning—as already indicated—can be diagnosed by the combination of clinical symptoms and laboratory findings. It is a gastroenteritis, which is accompanied by a fairly constant blood picture. A severe anemia is present, which in our cases was mostly of the hyperchromic macrocytic type. It is a hemolytic anemia due to the destruction of erythrocytes by the lead. There is a question as to the mechanism of the destruction. Cantarow and Trumper[9] feel that the lead increases the fragility of the erythrocytes so that they are less resistant to trauma.

As a result of the marked hemolysis there is a compensatory stimulation of the bone marrow resulting in a large number of normoblasts and reticulocytes (polychromatophilia) in the peripheral blood. All normoblasts and reticulocytes show the basophilic stippling which is so common in lead poisoning. In many cases even the mature cells show stippling. However, stippling in itself is not pathognomonic of lead poisoning. Anemia and stippling of red cells can be caused by agents other than lead. We are not relying on one laboratory finding, but on a combination of many, to reach a diagnosis.

Accompanying the anemia and stippling is a leucocytosis which often is very marked. There is a shift to the left in the non-segmented forms. No toxic granules are seen in the granulocytes. Platelets are usually increased. We believe the leucocytosis and thrombocytosis are probably due to the stimulation of the bone marrow caused by the anemia and are side effects rather than due specifically to the lead.

The urine usually contains albumin and in some cases one may see casts, and an increase in the urobilinogen, caused by increased blood

destruction. We have not seen bile in the urine except for a trace in case 1.

Leptospirosis has been suspected a number of times in the early stages of lead poisoning and the blood picture may appear similar to that of lead poisoning. However, in leptospirosis the number of normoblasts seen are never as numerous nor is their stippling of the normoblasts or red cells. The granulocytes sometimes show toxic granules. The thrombocytes are normal in number. In a large number of cases of leptospirosis, even if the animal is not visibly icteric, bile can be demonstrated in the urine.[10]

Uremia is another condition that may be confused with lead poisoning. The blood picture here shows a marked anemia at times but signs of replacement are usually absent. There is no stippling of the red cells. On the other hand, toxic granules in the granulocytes are seen, and usually more often than in leptospirosis.[10]

In no condition except lead poisoning do we find the same combination of clinical symptoms, laboratory findings, and response to treatment. In cases which have not absorbed too much lead and reach an irreversible stage, there is a dramatic response to parenteral and oral calcium. The ordinary infectious gastroenteridites do not respond to calcium therapy alone, and on the other hand, lead poisoning does not respond to any of the gastrointestinal protectives or antiseptics.

We are indebted to Dr. Harry Fisher of the Connecticut Agricultural Experiment Station for the chemical analyses.

Summary

1. We have described nine cases diagnosed as spontaneous lead poisoning in dogs.

2. Diagnosis was made from the clinical observation and laboratory examination.

3. A successful treatment is described.

4. Chemical analysis of the organs of two cases is given.

5. We feel that the incidence of this condition is much higher than a review of the literature indicates.

6. Any case of gastroenteritis, which does not respond to the usual treatments, should have its blood examined for a possible diagnosis of lead poisoning.

Addendum

After the foregoing was in type, another case was seen. A nine-year-old, female Pekingese had recurrent attacks of anorexia and abdominal pain. Physical examination revealed nothing of importance and treatment with intestinal sedatives was fruitless. Her blood was then examined and revealed the following:

Hemoglobin: 13.5 g.; R.B.C.: 4,970,000 per mm^3; Hematocrit: 42; Color index: 1.25; Mean corp. vol.: 85 per u^3; W.B.C.: 11,100 per mm^3; Polymorphs: 71%, Stabs: 5% Lymphocytes: 21%. Eosinophiles: 2%, Monocytes: 1%. Five normoblasts were seen for every 100 white blood cells counted; these and the erythrocytes showed toxic stippling. Some Howell-Jolly bodies were seen.

Urine was mailed away for chemical analysis and showed 2ppm. of lead; this is 40 times the maximum normal limit.

The dog is owned by antique dealers and she used to watch them remove paint from antiques with a high-speed brush which finely powdered the paint. This was done in a poorly ventilated corner of their cellar. Since the paint was very old, it probably contained lead and the dog inhaled enough of it to cause symptoms of poisoning. This is the only one of our cases where possible inhalation of the metal existed.

The owners were told the keep the dog out of the cellar and to ventilate their paint-removing area better. The dog was put on a high calcium diet and for eight months has been free of any symptoms.

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Interstate Rabies Control

Interstate action to check the spread of rabies was initiated by the states of New York, New Jersey and Pennsylvania in December, 1948, at a conference of officials of the three states, held in New York City. The conference was sponsored by New York's Joint Legislative Committee on Interstate Coöperation, headed by Assemblyman Harold C. Ostertag, and by the Council of State Governments.

Pursuant to a conference recommendation, the three states will coördinate interdepartmental efforts to control rabies, and within the states set up a tri-state continuing committee which will synchronize the rabies work in the region. New York has already coördinated its own rabies control program through an Interdepartmental Committee on which representatives of the Departments of Health, Conservation and Agriculture and Markets serve.

The tri-state action was taken as the result of an epidemic of rabies in central New York which has continued since 1944, and which affects a number of counties bordering on New Jersey and Pennsylvania.

Noticed first among dogs, the disease is now virtually under control in this species in New York as the result of mass vaccination programs and now the fox has replaced the dog as the chief vector of rabies. Up to mid-December of 1948, only 23 rabid dogs were reported for upper New York State for the year, but 244 rabid foxes have been recorded. Last year 52 rabid dogs were reported in upstate New York and 275 rabid foxes.

Cattle are the chief targets of New York's "crazy foxes", the conference was told. Farmers lost 440 cows in 1946 from rabies, 329 in 1947, and 210 in 1948. While these losses appear to be declining, both health and agricultural authorities believe it is due to the fact that the disease is now spreading in areas where dairy herds are more scattered, rather than to subsidence of the disease itself.

Pennsylvania authorities participating in the conference at which interstate control program was inaugurated, said that 22 rabid foxes were reported in 1946 in counties bordering New York's infected areas, but the incidence of the disease has apparently declined in Pennsylvania since then.

Dr. C. P. Bishop, director of Pennsylvania's Bureau of Animal Industry, cited the bounty system employed by that state as a possible factor in checking the disease among foxes. He pointed out that Pennsylvania paid out $158,697 in bounties on predators during the fiscal year ending May 31, 1948, and that the chief predators taken, numbering some 33,000, were foxes.

New Jersey authorities reported no incidence of rabies in foxes, as yet, but were alerted to the possibility of its appearance when New York authorities pointed out that many cases have been reported in Sullivan County, New York, on the New Jersey border. Dog rabies continues to be the prevalent problem in New Jersey, particular in Mercer, Somerset, Union and Middlesex counties, Dr. J. S. McDaniel of the New Jersey Health Department said.

New York State's efforts to check the disease in foxes have taken form of an intensive

In some areas the fox has to a large extent replaced the dog as the vector of rabies. Cattle are the chief targets of New York's "crazy foxes"

Courtesy American Foundation for Animal Health

trapping program carried on through the State Conservation Department by 27 professional trappers. Trapping has been done in 50-mile-square control areas where the disease has been most prevalent, and some 4,000 fox have been taken in these areas. A check is now underway to determine whether this method is effectively halting the spread of the disease.

John Wilson, coördinator of the rabies program for the New York State Conservation Department, expressed the belief, at the conference, that the professional trapping method would prove more effective than the bounty system, advocated by many sportsmen, inasmuch as the bounty hunters would not necessarily take fox from the infected areas and would not trap down the residual breeding stock as the professional trappers do.

Dr. Alexander Zeissig, veterinary consultant of the New York State Health Department, who has spearheaded the dog vaccination program for that state emphasized the importance of public education and voluntary cooperation in the mass vaccination programs as the most effective methods yet devised for combating rabies. He pointed out that compulsory vaccination programs, where instituted in other states, have not been successful* whereas, under the voluntary methods, correlated with a program of public education, from 70 to 90% of the dogs have been vaccinated in infected areas in New York with a corresponding reduction of canine rabies.

Sheep Parasite Investigations

Basic facts on internal parasites of sheep which often cause heavy losses to Western producers is being sought by research financed by the federal Research and Marketing Administration. With these facts it is hoped to develop handling methods that will prevent sheep from acquiring such parasites, as well as treatments for animals that are affected. Sheep diseases caused by parasites are more costly than any others to producers. Immediate objectives are to find out how the fringed tapeworm is transmitted to noninfested sheep, and also what parasite or parasites cause dysentery in lambs. Investigations in New Mexico indicate that the fringed tapeworm is not transmitted by contact with infected animals, but through an intermediate host. Experiments are under way to discover this host as well as other means of transmission. Studies at the Beltsville (Md.) Research Center indicated that the persistent scouring of lambs, which is especially common in the summer and fall in the Northern Plains, is caused by intestinal worms, although there may be other causes. Lambs placed in an infected pasture began scouring within three weeks and lost weight. But lambs placed on an adjacent, but clean pasture, remained healthy and gained weight. The project is directed by the Bureau of Animal Industry in cooperation with the experiment stations in New Mexico, South Dakota and Montana.

*Note.—This is not in accord with the record. Compulsory antirabies vaccination has been successful in every instance where enforced to the extent of 60% or more of the canine population.

Wheat Poisoning, Grass Tetany

A study of the cause and means of preventing death losses from grass tetany and other causes, in cattle and sheep pastured on grasses and small grains, especially wheat, is being made by the Bureau of Animal Industry, in cooperation with the experiment stations and other state agencies in Kansas, Oklahoma and Texas. The trouble generally is known as "wheat poisoning." For a number of years there have been severe losses in cattle and sheep pastured on wheat in these states. Animals become affected suddenly and may die within an hour. Losses in some years have run into millions of dollars. Prompt action by a veterinarian may save affected animals, but it is essential that the basic causes and means of prevention be determined, if losses are to be reduced or eliminated. Pleas from livestock men for research on "wheat poisoning" emphasize the seriousness of the problem.

Award to Doctor Fenstermacher

In recognition of his many contributions to the upbuilding of the state, Dr. R. Fenstermacher of St. Paul, Minnesota, president of the Minnesota State Veterinary Medical Society was presented with The *Minneapolis Star and Tribune* Award for Leadership in Minnesota.

The presentation was made at the 52nd annual convention of the Minnesota State Veterinary Medical Society in Minneapolis on January 4, 1949.

The award consists of a watch pendant inscribed "For Leadership in Minnesota" and a framed certificate which reads:

"In recognition of leadership in Minnesota. Whereas Dr. R. Fenstermacher as president of the Minnesota State Veterinary Medical Society has given a service to Minnesota that has added to this state's supremacy as a place in which to live and work, has brought additional respect to his organization and has enhanced his own prestige as a citizen, The *Minneapolis Star and Tribune* present to him this Leadership Award."

Sulfonamides in the Control of Experimental Coccidiosis in the Turkey*

By E. H. PETERSON, Ph.D., D.V.M.
Pullman, Washington

ALTHOUGHT extensive studies relative to the value of sulfonamides in the control of coccidiosis in the chicken have been conducted, no studies of the effectiveness of sulonamides in the control of coccidiosis in the turkey appear to have been made. The present study was undertaken to investigate the value of sulfonamides in the control of experimental coccidiosis in the turkey.

Methods

In order to assure a supply of poults for experimental purposes, eggs were secured for hatching purposes from a flock of 30 Beltsville Small White hens. Poults thus were made available in small lots over a considerable period. The newly-hatched poults were brooded from four to five weeks in metal brooders and then transferred to batteries for experimental work. They were utilized for experimental purposes between the ages of four and six weeks.

All testing was conducted with a single culture of coccidia, isolated a short time previously from a field outbreak of the disease. The organism was assumed to be *Eimeria meleagrimitis* since it was markedly pathogenic, causing lesions occurring mainly in the lower portion of the small intestine. Only one other species of coccidia, *E. meleagridis*, is described as parasitic in the turkey. This latter organism is stated to possess negligible pathogenicity.

In determining the minimal levels of sulonamides effective prophylactically against the test organism, the poults were exposed to the disease, and with the exception of experiments involving sulfaguanidine, were given simultaneously the sulfonamide dissolved in the drinking water. Sulfaguanidine was mixed in the mash. Medication was continued until no further deaths occurred in the controls. In most instances, losses began in the control birds on the 7th day following exposure to the disease and continued during the 8th and 9th days. With very heavy exposure, losses began as early as the 5th day, but with the major loss still occurring on the 7th day. During the course of the studies, the exposure dose of sporulated oocysts was increased, since the potency of the culture seemed to deteriorate with age.

Results

Observations regarding the minimal levels of drug intakes effective prophylactically against the disease exposure employed are recorded in table I.

From the data in table I, minimal levels of drug intake in the water effective prophylactically against the disease under the conditions of exposure employed were shown to be as follows: sulfaquinoxaline and sulfapyrazine, 0.005%; sulfachlorodiazine, 0.01%; sulfamethazine and sulfamerazine, 0.02%. The prophylactic level for sulfaguanidine given in the mash was 0.10%. Borax up to 0.2% in the drinking water was without effect.

In an effort to locate the critical period for medication, groups of poults were exposed to infection and subsequently medicated with 0.02% sulfamethazine in the water, the onset of medication being variable (table II).

From table II, it is seen that 100% survival occurred when medication was begun as late as 96 hours after exposure to infection. Mortality, however, occurred when the onset of treatment was delayed 120 hours or more after exposure.

In order to determine the minimal length of the medication period necessary for complete survival of infected birds, a second series of poults was exposed to infection with *E. meleagrimitis* and medicated with 0.02% sulfamethazine in the water, treatment beginning upon the 4th day following exposure, but with the length of the treatment period varying between the bird groups (table III).

From table III, it is seen that medication for 24, 48, or 72 hours, beginning on the 4th day following exposure, delayed but did not forestall mortality, losses occuring respectively three days after withdrawal of the drug in the first instance and four days after drug withdrawal in the third and fourth instances. Medication, however, for four full days beginning 96 hours after exposure prevented all mortality.

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* This investigation was supported in part by a research grant from the Lederle Laboratories of Pearl River, New York. Appreciation is expressed also to Merck and Company of Rahway, New Jersey, and to Sharp and Dohme, Glenolden, Pennsylvania, for supplying sulfaquinoxaline and sulfamerazine respectively.

To check these observations, a further trial was set up. Groups of poults were exposed very heavily to infection with *E. meleagrimitis* and subsequently given 0.04% sulfamethazine in the water for various intervals beginning 96 hours after exposure to the disease. Results are shown in table IV.

From table IV, 48-hour interval medication beginning on the 4th day after disease exposure delayed but did not prevent mortality when the interval following drug withdrawal exceeded 24 hours. In lot 38, where the drug was withdrawn for 24 hours only, loss was averted. Again, as is shown in table III, medication for four or more continuous days beginning on the 4th day after exposure prevented all mortality from coccidiosis. It should be noted that 100% of the controls succumbed to the disease.

Immunity Considerations

In order to test the development of immunity, a group of 15 poults were exposed to 600,000 sporulated oocysts of *E. meleagrimitis* and placed simultaneously upon 0.04% sulfamethazine in the water for 12 continuous days. At the end of that period, the birds were challenged with 1,200,000 sporulated oocysts of the organism along with five control birds. All five of the controls developed clinical evidence of coccidiosis although none of them died. The 15 previously-exposed birds were unaffected clinically by the challenge exposure. The results indicate that at least some degree of immunity against the disease may develop while the birds are being medicated with sulfonamide. The results are not to be regarded as conclusive, however, since the culture, even though it had been transferred recently through poults, had lost most of its original pathogenicity.

TABLE I.—PROPHYLACTIC ACTION OF SIX SULFONAMIDES AND ONE NON-SULFONAMIDE COMPOUND AGAINST INFECTION WITH *Eimeria meleagrimitis* IN THE POULT.

Lot	Poults	Oocysts per bird	Test Drug	Drug conc. in water	Death loss
1	10	200,000	Sulfaquinoxaline	0.0025	8
2	10	200,000	Sulfaquinoxaline	0.005	0
3	10	200,000	Sulfaquinoxaline	0.010	0
4	10	200,000	Sulfaquinoxaline	0.020	0
5	10	200,000	Controls		10
6	10	250,000	Sulfamerazine	0.010	0†
7	10	250,000	Sulfamerazine	0.020	0†
8	10	250,000	Sulfamerazine	0.040	0
9	10	250,000	Cintrols		7
10	10	250,000	Sulfamethazine	0.010	2
11	10	250,000	Sulfamethazine	0.020	0
12	10	250,000	Sulfamethazine	0.040	0
13	10	250,000	Controls		8
14	10	400,000	Sulfachlorodiazine	0.005	7
15	10	400,000	Sulfachlorodiazine	0.010	0
16	10	400,000	Sulfachlorodiazine	0.020	0
17	10	400,000	Sulfaguanidine	0.050*	3
18	10	400,000	Sulfaguanidine	0.100*	0
19	10	400,000	Sulfaguanidine	0.200*	0
20	10	400,000	Controls		10
21	10	400,000	Sulfapyrazine	0.005	0
22	10	400,000	Sulfapyrazine	0.010	0
23	10	400,000	Sulfapyrazine	0.020	0
24	10	400,000	Borax	0.050	10
25	10	400,000	Borax	0.100	10
26	10	400,000	Borax	0.200	10
27	10	400,000	Controls		10

* Sulfaguanidine given in the mash. †Clinically affected

Discussion

The prophylactic levels of six sulfonamide drugs against coccidiosis in poults due presumably to *E. meleagrimitis* were determined. It is well recognized, of course, that the prophylactic activity of a drug against coccidiosis varies with the degree of disease exposure. All exposures to disease in these observations were heavy since from 70 to 100% of the controls succumbed in each instance. This is a higher mortality rate than is experienced in most naturally-occurring outbreaks of the disease. It is felt, therefore, that the minimal prophylactic levels of the drugs in water as determined in these trials should be effective in dealing with field outbreaks of the disease. This assumes that other strains of *E. meleagrimitis* will not differ appreciably in their sensitivity toward sulfonamides, an assumption which may not be justified.

The critical period for medication is shown to be the four-day interval beginning 96 hours after disease exposure. All groups medicated at the prophylactic level of the test drug (sulfamethazine) over this critical period showed complete survival. With the exception of lot 38, all bird groups medicated for only a portion of the critical four-day period suffered mortality. It is suggested, therefore, that in field outbreaks of coccidiosis in poults, sulfonamide, if given, should be administered for a minimum of four consecutive days. Observations of actual field outbreaks alone can determine what additional medication may be necessary for the most complete suppression of such outbreaks of this disease.

Due to lack of poults, special toxicity studies

of the respective drugs at the prophylactic levels of intake could not be undertaken. No evidence of toxicity was observed, and it is probable that at least the commercially-available sulfonamides tested will be relatively innocuous to poults at the indicated levels of intake since these levels are far below those commonly recommended for the suppression of coccidiosis in the chicken.

The following tentative recommendations, however, are made for the control of field outbreaks. After an outbreak of the disease has been diagnosed by microscopic examination of intestinal contents, place the flock immediately upon a sulfonamide in the mash or water, but not both. If sulfaquinoxaline is the drug selected, use 0.02% in the mash or 0.01% in the water. For sulfamethazine or sulfamerazine, use 0.1% in the mash, or 0.04% in the water. Give sulfaguanidine, if used, as 0.21% of the mash.

Continue the medication until death loss stops. This should be within 48 hours. Then cut the medication one-half, *i.e.* if 0.04% sulfamethazine has been given in the water, reduce it to 0.02% etc. Continue medication at the lower level for a week to 10 days. Do not move the birds to other premises or change the litter, since it is desirable that they pick up oocysts and become exposed to the disease while they are being treated. These recommendations are tentative only.

TABLE II. MEDICATION OF POULTS WITH 0.02% SULFAMETHAZINE IN THE WATER FOLLOWING THE EXPOSURE OF EACH BIRD TO 250,000 SPORULATED OOCYSTS OF *Eimeria meleagrimitis*, THE ONSET OF MEDICATION BEING VARIABLE.

Lot	Poults	Date of Exposure	Beginning of Treatment	End of Treatment	Mortality
28	10	6/9	6/12	6/18	0
29	10	6/9	6/13	6/18	0
30	10	6/9	6/14	6/18	2
31	10	6/9	6/15	6/18	5
32	9	6/9	6/16	6/18	9

Conclusions

1. The minimal prophylactic levels of five sulfonamide drugs administered in the drinking water against *E. meleagrimitis* infection in the poult, using disease exposures that destroyed from 70 to 100% of the controls, were as follows: sulfaquinoxaline and sulfapyrazine, 0.005%; sulfachlorodiazine, 0.01%; sulfamethazine and sulfamerazine, 0.02%. The minimal prophylactic level of sulfaguanidine when administered in the mash was 0.1%. Borax up to 0.2% in the drinking water had no effect on the disease.

2. Bird groups exposed to infection and medicated at the prophylactic level of the test drug showed complete survival when medication was begun at any time up to 96 hours after exposure and continued for four days or longer, but mortality occurred when they were medicated for only a portion of this critical 4-day period. If medication was delayed for 120 hours after exposure, mortality followed. Therapeutic action of the sulfonamides against infection with *E. meleagrimitis* is therefore indicated.

TABLE III.—MEDICATION OF POULTS WITH 0.02% SULFAMETHAZINE IN THE WATER FOLLOWING EXPOSURE TO 250,000 SPORULATED OOCYSTS OF *Eimeria meleagrimitis* PER BIRD. MEDICATION BEGINNING ON THE 4TH DAY AFTER EXPOSURE AND VARYING IN DURATION BETWEEN THE GROUPS.

Lot	Poults	Date of Exposure	Days of Treatment Beginning 6/21	Mortality							
				6/24	6/25	6/26	6/27	6/28	6/29	7/1	Total
33	10	6/17	1		2		1				3
34	10	6/17	2				2		1	1	4
35	10	6/17	3					1		1	2
36	10	6/17	4								0
37	10	6/17	Controls	2	3	3	1				9

TABLE IV.—MEDICATION OF POULTS WITH 0.04 PER CENT SULFAMETHAZINE IN THE WATER FOLLOWING EXPOSURE TO 800,000 SPORULATED OOCYSTS OF *Eimeria meleagrimitis* PER BIRD, MEDICATION BEGINNING ON THE 4TH DAY AFTER EXPOSURE AND VARYING IN DURATION BETWEEN THE GROUPS.

Lot	Poults	Date of Exposure (August)	Periods of Medication (August)	Mortality									
				8/9	8/10	8/11	8/12	8/13	8/14	8/15	8/16	8/17	Total
38	10	4	8, 9; 11, 12										0
39	10	4	8, 9; 12, 13					2	5			1	8
40	10	4	8, 9; 13, 14			1	1	2		1			5
41	10	4	8, 9					1	1	3	1	1	7
42	10	4	8, 9, 10, 11										0
43	10	4	8, 9, 10, 11, 12										0
44	10	4	Controls	1	2	6	1						10

Avain Pneumoencephalitis (Newcastle disease) in Egg Laying Contest

By J. R. BEACH,* D.V.M.
Berkeley, California

THIS communication is a description of the outbreak of avian pneumoencephalitis (PE), or Newcastle disease, which occurred in the California Official Egg Laying Contest during the 1947-48 test year. It is presented as an illustration of the relatively harmless nature the disease has frequently assumed in the United States although in other countries it is reported as usually causing nearly a 100% mortality.

The chicken population at the contest consisted of 38 entries of 26 pullets each, a total of 988; there were two entries from each of six farms, and three and four entries respectively from each of two farms. Twenty-six entries were of California origin; 12 came from breeders in seven widely scattered states. The entries were confined separately in 38 pens, without yards, distributed equally in two houses (A & B). The pens were separated by solid wood partitions which prevented all direct contact between birds of different entries. The only access to any pen

Pullet with avian pneumoencephalitis. Note, leg and neck muscles affected

was by a door at the front. This was kept locked day and night; only caretakers being permitted to enter.

Careful inspection of each entry on arrival at the contest did not reveal evidence of the presence of any disease among the birds. October 28, four weeks later, a sudden and marked drop in the egg yield of pen 15, in house A, caused the presence of pneumoencephalitis (PE) to be suspected. A week later, November 3, pen 36, in house B, suffered a like decline in production. Between December 10, and January 10, three more pens in House A and two in House B were suspected of having become affected. A blood sample for hemagglutination-inhibition test (HI), a procedure used in the diagnosis of PE, was now taken from one bird selected at random from each of the seven suspected pens. The blood of the birds from six pens gave a positive reaction for PE and one was negative.

The next cause for suspecting the presence of PE was dullness, inappetence, and drop in egg yield of the birds in pen 33 during the latter part of March. HI tests of blood samples from this pen were negative. A companion entry from the same source in pen 32 which had not shown evidence of illness at any time, however, was tested and found positive for PE. These results indicated that the sickness in pen 33 was not due to PE and that pen 32 had a subclinical infection. At this time blood samples were taken from three other pens selected at random. Two of these were PE positive and one was negative. This brought the number of pens tested up to 12, of which nine were positive to the HI test for PE and three were negative.

September 28, two days before the close of the test year, blood samples for HI test were taken from three birds of each entry. The results of these tests for PE were as follows:

The blood samples from the three pens which had been negative and from the nine pens which had been positive to HI tests in January or March were, respectively, still negative and positive.

Of the 26 pens not previously tested for HI antibodies all of the three blood samples from each of 7 pens were positive, one of the three blood samples from each of 6 pens was positive, and all of the three blood samples from each of 13 pens were negative.

Thus the results of the HI tests show that all or part of the birds in 22 of the 38 pens at sometime had been infected with PE, but they do not indicate whether the infection occurred before or after the birds arrived at the contest. However, if one assumes that all birds coming from flocks which had gone through an outbreak of PE before the entries were selected would have been infected, it appears then that much of the infection detected by the HI test at the contest had actually resulted from spread from the two pens initially infected. This is shown by the fact that positive reactors to the HI test were found in only one pen of each of four of the six double entries, in only one pen of the

* Department of Veterinary Science on the Berkeley Campus of the University of California.

one triple entry, and in only two pens of the quadruple entry. The source of the infection at the contest is unknown. Two possibilities are: (1) one or more entries had undetected subclinical infection on arrival, and (2) chance introduction of the virus from the outside had occurred.

The fact that 16 of the entries appear to have escaped infection entirely shows that the segregation provided by the solid partitions between pens and/or the operational procedures followed had been effective to an appreciable degree in preventing the spread of a highly contagious disease.

The influence of the disease on the performance of the entries involved is difficult to evaluate because there is no certain means of estimating what their total egg yield would have been if they had not been affected. Comparison of records of the two pens of the double entries proved of little help. In two cases the performance of the infected pen exceeded that of the non-infected pen, and in one instance the positions of the infected and non-infected pens were reversed; in one instance in which both pens of a single breeder were infected their final places were the 15th and 30th. The egg yield of a majority of the infected pens declined temporarily, however, and two pens did not again lay at the preinfection rate. On the whole, it may be said, the loss in eggs from the effect of the disease did not appear to materially affect the total production for the test year. There were no deaths definitely attributable to PE although there were two deaths in one entry which might have been due to PE.

This mild outbreak of PE presents some unusual features. The most striking, perhaps, is that, on the basis of the results of HI tests, 16 of the 38 entries did not become infected. The complete separation of each entry from others provided by the solid doorless partitions between pens undoubtedly acted as a barrier to the spread of infection by direct contact between diseased and susceptible entries. This factor conceivably may also have been an obstacle to transmission of infection by the air-borne route between the pens and perhaps also between houses. One can only speculate on this point, however, because too little is known of the route of travel of air currents by which infection-laden dust would be carried. In no wise affected, however, was the possibility of mechanical transfer of infection by caretakers whose duties required they enter each pen several times daily.

Chick affected with pneumoencephalitis. Legs are paralyzed

Another somewhat surprising finding is the fact that, on the basis of the results of the HI tests, the infection in some pens did not involve all of the birds. This is quite contrary to the concept commonly held which is that although an outbreak of PE may cause no mortality the morbidity is likely to be 100%.

Displaying nervous symptoms of avian pneumoencephalitis

Observations of this outbreak concur with those of outbreaks on poultry farms in demonstrating that a flock of laying pullets can experience PE infection without any appreciable loss from any cause, resulting therefrom. They also show that it may be possible, although perhaps not practicable, for poultry raisers to provide housing facilities and adopt operational procedures which would enable them to confine any PE infection that may appear, to a small part of their flocks.

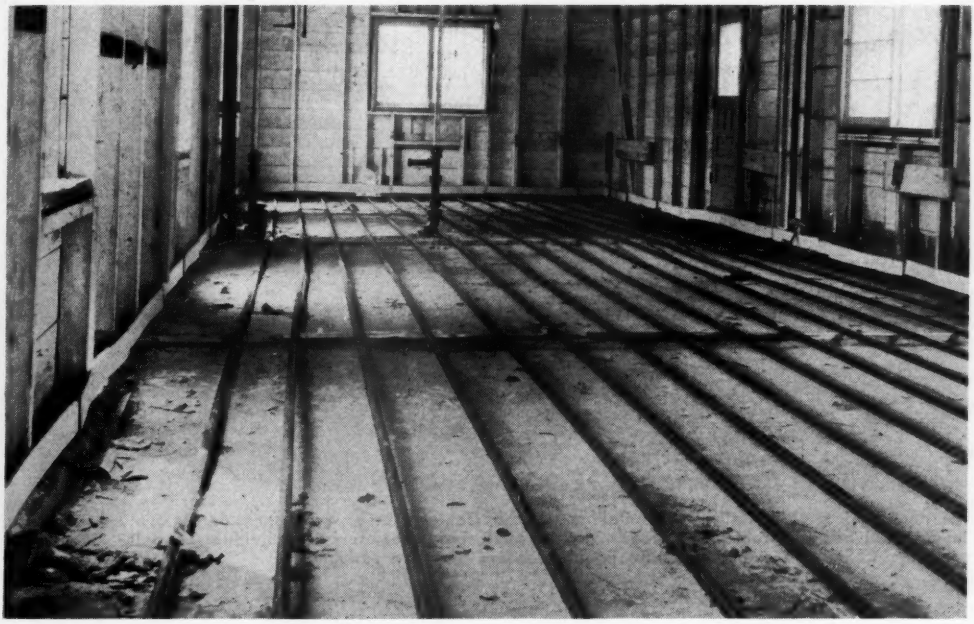
Radiant Heating for Animal Hospitals

As one of his first objectives after graduation in 1943 Dr. Joseph Beck planned to build an animal hospital. But in that year and for some time building materials were scarce, so for a year the doctor practiced from a rented residence in Massachusetts. Then came an opportunity to purchase a small piece of property near Newton Center, Massachusetts. Besides a residence, the property included an old hen-house, a barn and a few sheds. Doctor Beck pondered as to the possibility of the hen-house being made into an animal hospital. The location was perfect—near to a heavily traveled road and close to his residence. Visioning a modern hospital rising from the hen-house led him to consult a contractor who, after studying the situation pro and con decided favorably and set about construction of a veterinary hospital with the hen-house as the basis. Using what building materials were available in wartime the dirt-floored, dilapidated chicken house was converted into a modern, five-room hospital for pets. Among the up-to-date conveniences of the hospital is a radiant heating system.

This type of heating has been found by the doctor to be ideal for structures of this nature, inasmuch as concealment of the heating elements in the floor eliminates the hazard of frightened animals injuring themselves running against exposed, high temperature surfaces. The steady uniform heat at lower air temperatures and higher relative humidity that are provided by radiant heat achieves an excellent environment for healthy, as well as ailing, animals.

Radiant heat is based upon the principle of warming large surface areas to control body heat losses. Such systems eliminate entirely the need for exposed heating units and permit maximum utilization of space. Most commonly, radiant heating systems in houses, hospitals, churches, schools and virtually every type of structure, consist of wrought iron pipe coils or grids embedded in the floor.

When the floor slab is warmed, heat radiates to all areas of the building, including the tiers of kennels. With this type of heating system, drafts caused by convection currents are held to a minimum and heat is distributed uniformly. Temperature difference between floor



Wrought iron pipes welded together into a grid form the floor-type radiant heating element in this remodeled animal hospital. Positioning the grid on the dirt floor was the first step in converting the building from a chicken coop into a veterinary hospital*

*The A. M. Byers Company, Pittsburgh, 22, Pa., will supply information for the installation of floor, wall, or ceiling-type radiant heating systems.

and ceiling seldom varies more than one or two degrees.

This modest animal hospital includes an office, treatment and operating rooms, a lavatory, and a ward containing three tiers of individual stalls for small animals.

Remodeling the building began when the dirt floor was leveled and a wrought iron radiant heating grid was positioned on it. A concrete floor slab was poured to complete the heating installation by enveloping the pipes. Hot water is circulated through the pipe grid by a small pump controlled through an ordinary wall thermostat. A separate gas-fired boiler is used to supply the hot water for the hospital radiant heating system.

The system for the hospital was designed for a minimum outdoor temperature of zero F., and it has functioned excellently during coldest weather so far experienced.

Doctor Beck's hospital is by no means the only animal hospital equipped with radiant heating, among others that may be mentioned are the Mesa Veterinary Hospital, Mesa, Ariz., and one operated by Dr. L. E. Meckstroth, Sidney, Ohio.

This neat appearing veterinary hospital at Newton Center, Mass., was once an unlovely hen-house. The hospital has five rooms—the rear room, a ward, has three tiers of individual kennels. "Mac," the Collie boarder seems perfectly satisfied.

NEW HOSPITALS

Among the scores of veterinarians who have built new hospitals since the war the following may be mentioned:

John O'Harra, ICS'43
Merced, Calif.

A. L. Miller, CVC'18
Ashland, Ohio

K. B. Sherer, OSU'30
Fremont, Ohio

J. R. Bridenstine, OSU'40
Bowling Green, Ohio

E. C. Hackett, OSU'36
Youngstown, Ohio

D. S. Dimmerling, API'42 and Lucille Dimmerling, API'43
East Liverpool, Ohio

D. B. McBride, OSU'39
St. Clairsville, Ohio

D. F. Blackford, OSU'46
Loraine, Ohio

Robert L. Burch, WASH'43
Seattle, Wash.

Earl Kennedy, TH'15
Moline, Ill.

W. G. Sessions, ICS'42
Burlington, Iowa

Max W. Harry, ICS'43
South Pasadena, Calif.

B. V. Allen, ICS'34
Ft. Wayne, Ind.

L. L. Boxwell, ICS'40
Cedar Falls, Iowa

Gordon C. Kendall, API'28
Montgomery, Ala.

D. K. Christian, KSC'42
Moorhead, Minn.

Clinical Reports

Infection in a Heifer Suppressed With Penicillin

January 4, 1949, a call was answered to see a good, dairy heifer, 29 months of age, with a long, large abscess in the region of the gastrocnemius. She was a gentle, 4H-Club, heifer, due to calve and had already developed a fine, large, well-formed udder. We handled her with great care to avoid bruising the udder. We placed her in a stanchion, a bull lead in her nose and a lariat around her fetlock, with the other end wrapped around a stanchion post, and the owner holding both ropes. By one stroke with a pointed curved bistoury, an incision about four inches long was made deep into the abscess. Seemingly about one-half gallon of pus was released and ran into the gutter. The cavity of the abscess was syringed out with warm water containing aseptogen, and followed by an irrigation with an iodine solution. The heifer made no struggle; did not even move. Apparently she felt no pain.

The weather was near zero and for fear the edges of the wound, which could not have had a good blood supply, would freeze, she was placed in a hay barn with bales piled 10 feet high on three sides of her and plenty of good bedding to lie on. It made a fine place for her in such cold weather.

The second day after the abscess was opened the owner called me to see the heifer. I answered the call expecting an obstetrical case, but to my surprise it was a case of a very different kind.

The following is what I found. The animal had been moved into the milking barn and put in a stanchion before I arrived. The wound, where the abscess had been, looked fine and was not swollen, but the heifer had a swelling that extended from the vulva down and forward to the ziphoid region. It was about 16 inches wide and included the udder. There was a small slough on the lower end. The teats were swollen, hard and icy cold. A straw-colored fluid was oozing through the skin of the teats and lower part of the udder, and dripping onto the concrete floor constantly. The exudation in time wet the floor. The heifer's temperature was 98°F. She had a constant, forlorn, distressing moan; the eyelids were slightly puffed, and she would not eat anything.

Wanted: a diagnosis.

My diagnosis was: Generalized infection with metastasis, and onset of gangrene of the udder.

My prognosis was: death.

She was the boy's 4H heifer and both he and his father insisted that she be treated. I yielded. Treatment and care was as follows:

Crystalline penicillin G potassium, 200,000 units intramuscularly and a digestive tonic per os, and a stock preparation of aloes and sorghum molasses, with yeast added at the time of giving. The udder and the entire swelling was bathed and massaged with an antiseptic oil. The heifer was then returned to the hay barn. Instructions were given to bathe and massage the udder and give a dose of medicine every two hours until I returned, the next day.

The next forenoon to my surprise the heifer was better. She was again given 200,000 units of crystalline penicillin G potassium, intramuscularly. Instructions were given to continue the same treatment as before.

The third day to my surprise, the heifer seemed to be normal in every way. She was eating alfalfa hay like any normal cow. The swelling was nearly all gone, even the swelling of the udder was gone and it was only about three-fourths as large as it was 36 hours previously. She was again, for the third time, given 200,000 units of crystalline penicillin G potassium, intramuscularly, with instructions to continue the other treatment twice a day.

At 11 a.m. the same day I was called back to see the heifer again. She had already given birth to a normal calf, shed the afterbirth and was standing up chewing her cud and the calf was trying to nurse, when I arrived. The milk was white and looked normal. The heifer and calf also looked normal.

I have never been sold on penicillin as a cure-all, not even for mastitis, but some times to my surprise, it seems to work a miracle, especially in conjunction with other indicated treatment. It seemed to do wonders here. The heifer and calf are both doing well.

S. L. STEWART, D.V.S.

Olathe, Kans.

Hernia Operation in a Pig

Over the years I presume I have done most of the "trick" operations described in the literature for umbilical hernia in shotes. Result: As far as I remember the pigs recovered but most of them included so much fussy trimmings that either the owner of the pig couldn't afford to pay the bill, or considering the value of the animal I couldn't charge a reasonable fee for the surgery.

One night last spring a client drove into my place bringing a pig eight weeks of age with an umbilical hernia of the size of my fist. Assisted by the owner I operated on the ground by the light of his truck. With the shote on his back, the intestines were easily

returned to the abdominal cavity, since the hernial opening was of about the size of a quarter-dollar. Although easily accomplished this part of the operation was painstakingly performed, just the same, to make certain all intestine was out of the hernia. Then two deep mattress sutures with strong suture material tied taut completed the bloodless and nearly painless operation in about the time it has taken to read this classical description of it.

Oh yes! Recovery was uneventful. That is, so far as the pig was concerned, it recovered promptly without unfavorable sequel; but for myself, I'm just waiting to do four more such operations with the same result, before giving this method a short, snappy name, probably "The Morgan Simplified Operation for the Relief of Porcine Umbilical Herina."

REMBRANDT MORGAN, D.V.S.
Winfield, W. Va.

Occurrence of Cytoleichus Nudus in Pheasants

Order: Acarima
Family: Cytoleichidae
Genus: Cytoleichus
Species: Nudus
Hosts: Fowl, and pheasants
Locality: Zoological Gardens, Lahore, Pakistan

Cytoleichus nudus (Vizioli) occurs in the air passages, lungs, liver and other organs of the fowl, turkeys, etc. Dickson and Lewis (1939) state that in serious infestations in the fowl, unthriftiness, coughing, "going light," paleness of head and dryness of feathers are noticed. When in large numbers the mites irritate the lungs and predispose them to pneumonia and pulmonary tuberculosis. Turbet (1929) states that these mites are responsible for causing a certain percentage of mortality in fowls in Fiji Islands.

The object of this note is to record the occurrence of *C. nudus* in three, aged pheasants (Lady Amherent, Silver and Golden pheasants) and one fowl that died from various diseases in the Zoological Gardens, Lahore; except that in the case of one aged pheasant, the heavy infestation of mites was the probable cause of death.

The pheasants were kept in the Zoo in one cage. Their ages varied from 14 to 16 years. They were purchased at Calcutta when they were four or five years old. On post-mortem examinations the mites were seen as white specks on the peritoneal surface, on the liver, kidneys, etc. They were not seen in the bone marrow of the long bones. Microscopically these mites were identified as *Cytoleichus nudus*. A number of hairs were seen on the legs and body of the mites.

References

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Herms, W. B. (1946) Med. Entomology, 491, Macmillan Co., New York.
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M. MAQSOOD
Punjab Veterinary College
Lahore, Pakistan

Carcino-Liomyoma in Turkeys

Carcinoma of the female genitalia in poultry, primarily originates in the ovaries or oviduct and further it gives rise to multiple and often widespread transcelomic implantations in other organs. The tumor though malignant, has little or no tendency to form true metastasis. From Jackson's (1936) discussions on the subject, it appears that much confusion surrounds the problem of histogenesis and classification of the tumor and the reasons suggested by the author are in favor of classifying the tumor as carcinoma liomyomatosum (liomyoma). Various other nomenclatures i.e. adenocarcinoma adenoma fibroids, liomyo-carcinoma etc., had been adopted by other workers.

This note is written with the object of recording three cases of carcinoma of the ovaries in turkeys, post-mortem examinations were performed on four turkeys (three female and one male) which were slaughtered for food purposes at Lorang's, Lahore. The carcasses were in fair condition. The ovaries in the female turkeys presented a much lobulated growth, consisting of multiple pedunculated and confluent nodules, greatly varying in size and shape. Each growth was about the size of a human fist. The smallest nodules were irregularly spherical and were about 0.5cm in diameter while the larger ones were somewhat oblong, varying from 2 to 3cm in diameter. The growths were yellowish white in color and a few smaller ones also presented small bluish areas. They were firm and sections of the nodules presented smooth surfaces. Microscopic characters of the tumor resembled those of the so-called carcinoma liomyomatosum.

Reference

Jackson, C., 1936. *Onderstepoort, J. Vet. Sci.* 6:92-113.

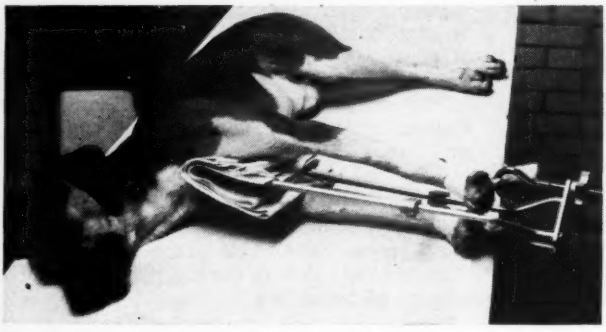
M. MAQSOOD
Punjab Veterinary College
Lahore, Pakistan

Treatment of Dislocation of the Elbow in Dogs*

THE ELBOW of dogs is subject to dislocation, and patients so affected are presented for treatment. Reduction in most cases can be annoying due to contraction of the muscles and the anatomical make-up of the bones forming the joint. Stader[1] described a method of reduction wherein a pin or Kirschner wire is inserted through the olecranon; to this is attached a pin-spreader. A retractor is used

Radiograph of a dislocated elbow joint

by placing the crutch-like head against the axilla and by means of a chain, attach the pin-spreader to the sliding hook. Steady traction is applied. The elbow is forced into extreme flexion during this procedure and by normally affecting a rotation movement of the forearm, reduction can be effected.



The Gordon extender is applied to the affected leg until the involved muscles are fatigued

Method of Reduction

The Gordon extender is of particular value in accomplishing reduction in these cases. The animal is anesthetized with phenobarbital sodium. The Gordon extender is applied in the prescribed method by placing the crutch end in the axilla and then attaching the unaffected leg to the stationary hook and the affected leg to the adjustable hook. Gradual

Lateral view of a disarticulated joint

traction is applied for about 15 minutes. Gentle kneading of the muscles and ligaments in the region of the elbow aids in accomplishing relaxation. After the soft structures are fatigued and relaxed the Gordon extender is removed, the elbow is placed in extreme flexion, and by rotating the forearm reduction is accomplished.

If one is not familiar with the contours of the bones making up articulation, a review of the anatomy aids considerably in handling these dislocations. The majority of cases require no after-treatment. In the few cases requiring after-treatment, to eliminat the possibility of recurrence, the joint is immobilized in the extended position by the use of the Thomas splint or by the application of a cast. The period of immobilization depends on the severity of the injury; usually, several days to a week is sufficient. One case of recurring dislocation was treated successfully by first accomplishing reduction, then the soft tissues surrounding the joint were infiltrated with pin-point injections of 50% Lugol's solution, and the leg was splinted with the elbow in the extended position for two weeks.

Summary

A method of accomplishing reduction of dislocation involving the elbow joint is presented.

Reference

Stader, Otto. Some Orthopedic Problems. *N. Am. Vet.* 20:62-66 (August) 1939.

By W. O. BRINKER,† D.V.M., M.S. and E. K. SALES,† D.V.M.

East Lansing, Michigan

*Presented at the 19th annual meeting of the Florida State Veterinary Medical Association, Oct. 24-26, 1948, Orlando.

†Michigan Agricultural Experiment Station, Section of Surgery and Medicine.

An Attempt to Infect Mink and Fox with Erysipelothrix Rhusiopathiae*

Hartsough[1] first isolated *E. rhusiopathiae* in ranch-raised mink. The isolation was made from a young kitten which showed a loss of appetite as the predominating symptom. In another case in which the cause of death was obscure, he recovered the same agent.

Many mink ranchers both in the United States and Canada have considerable difficulty raising mutation Aleutian mink. This particular color phase, although very beautiful, is susceptible to a variety of conditions, because they probably have a genetic constitutional weakness. We recently received four of these mink from a rancher in western Oregon. He had lost most of his Aleutian mink and the

remainder were dying. From the heart blood of one of these mink, we isolated an organism which conforms to the biochemical characteristics of *E. rhusiopathiae*. Since the heart blood and spleens of the other three mink showed no growth, we feel that the isolation of *E. rhusiopathiae* was incidental to the underlying cause.

This is the only instance in which we have recovered the agent from either mink or fox on routine bacteriological examination here at this station. According to the available literature, no isolations have been made from fox.

* * * *

In order to test the pathogenicity of the erysipelas organism, Hartsough[1] inoculated a series of mink intramuscularly, subcutaneously and intraperitoneally. None of the mink showed any symptoms, while his control pigeons succumbed. Since *E. rhusopathiae* infected pork and fish is probably being fed to fur bearers in some instances, we repeated the work done by Hartsough to further check the pathogenicity of the organism.

We obtained a culture of *E. rhusiopathiae* that had been isolated in a natural outbreak in turkeys the previous week. After isolation, the organism was grown in serum broth for 20 hours at 37.5° C. It was then inoculated intramuscularly in 1.5cc amounts to four healthy mink, intraperitoneally in 1cc amounts to the same number and fed in 50cc portions to four additional mink in their regular diet.

Foxes were simultaneously exposed in the same maner. Two foxes were given 2.5cc intramuscularly, two were given 2cc intraperitoneally and two were each fed 100cc in their regular diet. At the end of 40 days, no symptoms were noted in any of the animals, while control mice were killed in less than 40 hours and control trukeys in less than four days.

From our observations and those of Hartsough[1], we feel that the occasional occurrence of *E. rhusiopathiae* in pork and fish scrap probably will not cause disease in healthy mink and fox.

JOHN R. GORHAM, D.V.M., M.S.
Pullman, Washington

* From the Fur Animal Disease Research Laboratory, U. S. Bureau of Animal Industry in collaboration with the Division of Veterinary Science, Agricultural Experiment Station, Washington State College.

[1] Hartsough, G. R. 1945. Isolation of *Erysipelothrix Rhusiopathiae* from farm-raised mink. *J. Am. Vet. Med. Assn.* 107:242-243.

* * * *

Suppurative Nephritis in Mink*

Suppurative nephritis is a condition that is occasionally found when autopsying mink. It causes the death of probably one or two animals each year on a large ranch.

The malady is not considered contagious. It usually appears in mature mink and males and females are equally susceptible. The cause is bacterial and a variety of organisms have been incriminated.

Symptoms are usually absent. According to the owners, the animal had a normal appetite, but was found dead, with no previous history of being sick.

On post-mortem examination, the most significant lesion is a slightly enlarged soft, light-colored kidney. When the kidney is incised and its glandular surface exposed, the tissue appears very light in areas and at the pelvis a small amount of greenish-yellow pus is noted. A calculus may or may not be present, and one or both kidneys may be affected. The spleen is often enlarged and pale.

Since the disease is difficult to diagnose in the live animal, and is usually discovered only at autopsy, treatment is usually impossible.—JOHN R. GORHAM in *Fur of Canada* 14:33 (February) 1948.

Abstract

Quinacrine Hydrochloride in Tularemia and Brucellosis

During the past three years Ecke[5], observed eight patients with typical symptoms of ulcero-glandular tularemia. Every case was traced to handling rabbits a few days before. The patients were given a supply of 1.5 grain (0.1 gm.) quinacrine tablets with instructions to take one tablet three times a day for five days. Iodized ointment was prescribed for external application to the ulcers. Without exception quinacrine produced favorable results within one to three days, and no patient required treatment after the fifth day. These favorable results induced the senior author to try medication with the same dose of quinacrine hydrochloride (one tablet of 1.5 grains three times a day, for five days) in four patients infected with *Brucella abortus*. Here again the results were encouraging in that a recurrence developed in only one of the four patients after an interval of several years. Either streptomycin was not available or the patients could not afford it.

* * * *

Cataplasms for Acute Dermatitis in Horses and Cattle

The usual local treatment of canker and rope burn in horses and of foot rot and foot abscess in cattle is notoriously unsatisfactory, principally because of the long period required for recovery.

In a recent article Rainey[10] suggested that copper sulfate, zinc sulfate, iron sulfate, nitric acid, formalin, powdered alum, zinc chloride, butter of antimony and like caustics be replaced in the treatment of this condition with bland medicaments of which he finds mixtures of Fuller's earth and glycerine, such as cataplasma kaolini or antiphlogistine, the most satisfactory.

The cataplasm is applied cold in a thick layer to the skin around the affected part, in cattle particularly to the skin in the interdigital space, and renewed two or three times daily. No bandage is used. Few cases required a second visit from the veterinarian.

Except in advanced cases where there was extensive necrosis as a result either of neglected or maltreatment with severe caustics, Rainey's cases were speedily relieved by an application of antiphlogistine and recovery was rapid and uneventful. Formerly under severe forms of treatment speedy relief was exceptional and resolution often took months.

To quote the author's "discussion"—

"It had been long known that the cold kaolin-glycerine mixture was the best available means for treating acute inflammation of the skin in the vicinity of the foot of the horse. The condition known as 'cracked heels,' which is so common in army practice as a result of the horse getting his foot over a head rope or the picket line, is much more satisfactorily treated by kaolin-glycerine than by any sort of ointment.

"Indeed some equine skins are irritated by the fatty acids in an ointment based on fats, and even by a paraffin base.

"It was, consequently, reasonable to conclude that a similar safe and beneficial softening of the skin in foul-in-the-foot of cattle could be effected better by the kaolin-glycerine mixture than by other means.

"In both 'cracked heels' (rope burn) in horses, and 'footrot' in cattle, the gravity of the secondary pathological changes is due to the tension of the skin where it is normally tightly stretched in the vicinity of the horn of the foot.

"The extreme tension causes a local necrosis and the resultant sloughing exposes the underlying tissues to bacterial invasion. A rational approach to treatment thus primarily requires that the extreme tension of the inflamed skin must be relieved by a controllable and safe means. It is considered that a vegetable poultice does not fulfil these conditions because, if long continued, it causes maceration and incubates bacterial life to a dangerous extent, thus being apt to do more harm than good.

"A cold kaolin-glycerine application does not macerate the skin, and probably bacterial activity is diminished by the presence of the glycerine.

"Observation has proved that the early relief of skin tension given by kaolin-glycerine obviates local necrosis and thus prevents infection of tissues exposed by the separating slough. Consequently it should be the best means to use, and the results obtained have justified this assumption.

"Photo-sensitization, in a very severe form, is sometimes observed in Tasmania. Prompt treatment is necessary to prevent extensive necrosis of the skin. In these cases, and in other acute inflammatory conditions of the skin of the teats and udder, a better response

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[5]Ecke, D. H., 1948. Tularemia and undulant fever treated with atabrine. *Illinois Med. J.* 98:318. (June) Abst. in *J. Am. Med. Assn.*

[10]Rainey, J. W., M.R.C.V.S. 1948. Treatment of foul-in-the-foot and acute inflammation of the skin by a kaolin-glycerine mixture. *Australian Vet. J.* 24:164 (July).

follows the application of cold kaolin-glycerine, frequently renewed, than ointments based on fats or paraffin."

Aureomycin Therapy in Human Brucellosis Due to Brucella Melitensis

In 24 patients[2] with proved acute and chronic brucellosis due to *Brucella melitensis*, the immediate therapeutic results obtained with orally administered aureomycin surpassed those obtained with any other specific therapy (including a combination of streptomycin and sulfadiazine). This report is being made before a follow-up study, because the immediate results were so encouraging and so unexpected. Comparative studies *in vitro* and in experimentally infected chick embryos had shown that, weight for weight, streptomycin is more active against the Brucella of all three species than is aureomycin.

Aureomycin produced mild toxic effects. When small initial doses were used, the febrile reaction and drop in blood pressure, frequently observed with larger initial doses, were less severe and less frequent.

Further bacteriologic studies were carried out over a period of one to three months following therapy. A total of 82 cultures of blood from the 24 subjects have been studied. In three patients, one of four cultures taken after treatment showed *Br. melitensis*. The dose now recommended is four to six grams per day for two weeks instead of a total daily dose of two grams.

Typhoid Bacilli and Milk

A cow given, by the mouth very large doses of virulent typhoid bacilli (275 and 125 billions) on two occasions at an interval of 29 days failed to show signs of infection or to pass the bacilli in feces or milk[4].

Of two-month-old calves, dosed with about 100ml ascites broth culture of typhoid mixed with milk, one became infected and typhoid bacilli were grown from scrapings of the intestine and ileocecal gland. Both, however, passed typhoid bacilli in the feces on the first or second day after dosing.

When large numbers of typhoid bacilli (say, one to three billion) were instilled into the udder through the teat canal, excretion of the bacilli in the milk usually ceased within 10 days (14 quarters of four cows). In two quarters of one of these cows excretion continued for 25 and 27 days, and in two quarters of another cow for at least 85 days. The procedure usually caused an acute mastitis lasting for a few days, and the "H" and "O" agglutinin titer of the blood rose.

When large numbers of typhoid bacilli were applied to the teat orifice, even when this had been grossly injured beforehand by slitting the sphincter, there was as a rule only a very transient infection. In 11 of 12 of the cases, either the organism could not be found in the milk or was found for one day only; in the remaining case there was intermittent excretion for seven days.

These experiments do not support the view that milk-borne typhoid outbreaks are brought about by an active infection of the cow and suggest that the main object of the epidemiologist must still be the location of the human carrier.

Lameness in Sheep Due to Swine Erysipelas Infection

In the past few years considerable loss has been experienced in New Zealand sheep following dipping, from loss of condition consequent upon lameness and difficulty in getting about to graze.[11]

Dipping in freshly prepared dip occasioned no difficulty, but, after being used and standing for several days or weeks dip sometimes infected 20 to 80% of the sheep subsequently immersed in them causing slight to severe lameness. In the main there were no lesions, but, upon necropsy, some of the more severe cases showed moderate swelling of the coronet and some edema and congestion of this region. In most instances slight cuts, punctures, abrasions or other minor wounds of the fetlock suggested the portal of microbian invasion.

A recent study[12] has shown this lameness in sheep following dipping to be due to infection with *Erysipelothrix rhusiopathiae*. Affected animals improved when given penicillin, but not spectacularly. Three doses of penicillin given before the symptoms developed prevented the disease in animals inoculated experimentally.

It was found that infection of the sheep from dipping could be prevented by adding bacteriostatic substances to the dip. The addition of copper sulfate, three pounds per 1250 gallons of dip, is effective in preventing the post-dipping lameness and is recommended, also, for benzene hexachloride and derris (rotenone) dips.

[2]Spink, Wesley W. and Braude, Abraham I. Div. Ins. Med., U. of Minn. Hosp. and Med. Sch.); Castaneda, M. Ruiz and Goytia, Roberto Sylva (Mexico Gen. Hosp., Mexico D. F., Mexico). *J. Am. Med. Assn.* 138:1145-1148 (Dec. 18) 1948—J. L. Davidson.

[4]Minett, F. C., 1947. Experiments on infection of cows with typhoid bacilli. *J. Hygiene* 45, (May). Author's summary.

[11]Dipping lameness in sheep. *Vet. Med.* 43:495 (December) 1948.

[12]Cutaneous Erysipelothrix infection in sheep. *Australian Vet. J.* 24:157. (July) 1948.

AVMA 14th International Veterinary Congress Tour

The AVMA European tour planned for those who wish to visit other countries in connection with the 14th International Veterinary Congress in London from Aug. 8 to 13, 1949, is now definitely arranged as to places that will be visited and the time to be spent at each place. Complete information as to space on transatlantic boats is not yet available. This leaves the matter of price somewhat undetermined. The minimum cost of the tour by steamship (cabin class) will be approximately $1,450. However, not all space in any class carries a minimum rate. The price depends, in part, on the location and the number of persons assigned to a stateroom. The round-trip rate of the tour (first-class) on "queen" steamers, or by plane, will range upward from the minimum approximately $250.

Plans of the steamships, showing the space available and the rate, will be mailed as early as possible to those making reservations, so that the choice of space may be indicated and also, whether the transatlantic crossing by air or surface craft is preferred. Full information concerning luggage, passports, mailing addresses, etc., will be mailed at an early date to those making reservations.

The maximum space available to the AVMA party includes accommodations for 70 persons. This is fewer than attended either the 1930 or the 1938 Congress, from this country, but it is all the space that could be procured even though application was made last June. Not all of it has been reserved as yet. Those planning to make the tour, but who have not made reservations, should do so immediately. A deposit of $450 per person is required with reservations. Reservations may be cancelled up to 30 days before the sailing date with full refund of the deposit.

The schedule of countries, cities, and places that will be visited on the tour is as follows:

Itinerary for Tour of Europe—1949

July

15 Sail from New York on "S.S. Queen Elizabeth."

Courtesy Allied Laboratories, Inc.

16 to 19 At sea.

20 Due to arrive Southampton. Continue by boat train to London.

21 Leave London by morning train for Harwich continuing by night steamer.

22 Arrive Esbjerg, Denmark, at 9:00 a.m. and continue by train to Copenhagen, arriving about 3:00 p.m.

23 In Copenhagen, City sight-seeing drive including the Old Town, the Charlottenborg Palace, Thorwaldsen Museum, Rosenborg Castle, Tivoli Gardens, and other places of interest. Visit the Royal Veterinary and Agricultural College and the State Serum Laboratory.

24 Train to Stockholm.

25 In Stockholm. Morning of sight-seeing in the city visiting the Royal Palace, Riddarholmen Island, the New Town Hall and

other important places. Afternoon, visit the Royal Veterinary College and the State Veterinary Bacteriological Institute; motor trip to Drottningsholm, the "Swedish Versailles."

26 Train to Oslo.

27 In Oslo. Morning of sight-seeing, visiting Akershus Castle, Royal Palace, Open Air Museum with its Norse farmhouse and ancient viking ships.
Afternoon visit State Veterinary Institute.

28-29 Overland tour of Norway fjords *via* Myrdal, Flaam, Stalheim, and Voss.

30 Arrive Bergen about noon and sail B & V steamer about 2:00 p.m.

31 At sea.

August

1 Arrive Newcastle-upon-Tyne and continue by train to Edinburgh.

2 In Edinburgh. Morning of sight-seeing, visiting the Royal Castle, St. Giles' Cathedral, John Knox House, the Royal Mile, Princess Street, Holyrood Palace, and Abbey.
Afternoon, visit the Royal (Dick) Veterinary College and Moredun Research Institute.

3 Full-day excursion by motor coach through the Trossachs, visiting Stirling, Bannockburn, Loch Lomond, Loch Katrine, etc.

4 Excursion to Abbotsford, Dryburgh Abbey, Melrose, Glamis Castle (Macbeth's home), etc.

5 Train to English Lake District.

6 Motor coach tour to the Lake District, visiting Windermere, Keswick, Grasmere, etc.

7 Morning train to London. Arrive before 5:00 p.m. (Certain social functions in evening.)

8-14 In London. Attend the 14th International Veterinary Congress. (Transfers to and from Congress not included.)

15 Visit Ministry of Agriculture and Fisheries, Veterinary Research Laboratories at Weybridge, etc.
Leave London in the evening by train for Harwich, continuing by night steamer to Holland.

16 Arrive at the Hook of Holland in the morning. Visit Schiedam abattoir, the newest and most up-to-date in Europe, and/or Rotterdam, thence to Amsterdam.

17 In Amsterdam—largest city in Holland. Visit the celebrated Ryks Museum, containing many Rembrandt paintings, the Royal Palace, diamond cutters, and other places of interest. Also, half-day excursion to quaint Volendam and the Isle of Marken.

18 Train to Brussels.

19 In Brussels—the "Little Paris," capital of Belgium. Motor tour of the city, visiting the Grande Place, Hotel de Ville, Palace of Justice, Cathedral, and the unique Wiertz Museum. Visit l'École de Médecine Vétérinaire de l'État.

20 Train to Basle.

21 Morning train to Lucerne.

22 and 23 In Lucerne—"queen of Swiss resorts." Excursion by lake steamer and mountain railway to the summit of the Rigi.

24 Motor coach to Interlaken *via* the Furka and Grimsel passes and the Rhone Glacier.

25 In Interlaken, at the foot of the mighty Jungfrau. Excursion by mountain railway to Scheideck, *via* Lauterbrunnen and Wengern, returning by way of Grindelwald.

26 Morning train to Berne. Half-day sightseeing in this remarkably preserved medieval city, including the veterinary college.

27 Train to Paris.

28 to September 1 In Paris. One day of sight-seeing in the city, visiting the Louvre, Notre Dame Cathedral, Napoleon's Tomb, the Tomb of the Unknown Soldier, and the principal boulevards and buildings. On another day, motor to Versailles, visiting the Palace and gardens, also the Chateau of Malmaison, and a half-day visit to l'École National Vétérinaire d'Alfort.
One evening trip to night clubs.
Three free days for shopping, preparing for return journey, etc.

2 Fly Pan-American Airways.

3 Arrive New York.

Or by boat and reach New York six days later.

To repeat: (1) Some space is yet available on this tour. (2) The price ranges from approximately $1,450 to $1,700, depending upon the ocean transportation (surface or air) and the price of the stateroom. (3) The space is mainly cabin class but a few first-class reservations are available. The "Queen" steamers are the finest boats afloat and cabin class on them is superior to what first-class was on any boat only a few years ago. (4) The price of the tour includes transportation from New York and back to New York; all scheduled sightseeing trips; hotels and meals, except lunch and dinner during the Congress; gratuities, except tips on the boats; conveyance of baggage; and admissions to museums or other places where a charge is made. (5) The hotels which will be patronized in Europe are classed as "Ol" and are comparable to the La Salle Hotel or the Sherman Hotel in Chicago. (6) Those making the eastward transatlantic passage by air will leave New York on July 19 and join the main party in London. (7) September sailings are not definitely set at this writing, but those returning by boat will probably leave Cherbourg September 2 or Southampton September 3 on a Transatlantic liner and arrive in New York six days later.

Reservations, accompanied by a deposit of $450, should be sent to Dr. D. M. Campbell, 7632 S. Crandon Ave., Chicago 49, Ill.